VOLUME 68 • DECEMBER 1958 • NUMBER 6

A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

INDEX NUMBER

Myocardial Functional Failure Occurring in Respiratory Obstruction
James F. Gardner

Stapes Mobilization
Edward H. Campbell

Growth of the Nose and the Septorhinoplastic Problem in Youth
Joseph G. Gilbert and Samuel Segal Jr.

Peristapedial Pathology and Otitic Reinfection
George Kelemen

Recruitment Measured by Automatic Audiometry
Bernard A. Landes

Subcutaneous Emphysema
Burton R. Andersen

Nasal Sinus Disease
Henry M. Goodyear

Intranasal Chromophobe Adenocarcinoma
Lawrence J. Lawson

Associated Carcinoma of Larynx and Lung
Alan S. Rubenstein, James Graham, and Neale Barnes

Cerebral Mucormycosis
Howard W. Smith and John A. Kirchner

Tracheostomy
John M. Loré Jr.

Secretory Otitis Media in Children
Pierce W. Theobald

Secretory Otitis Media and Nasal Allergy
Don J. Weekes

The Education of a Young Deaf Child Begins with His Parents in His Home
Mrs. Spencer Tracy

A Rapid and Simple Technique for the Detection of Nonorganic Hearing Loss
Dean A. Harris

Combined Internal-External Laryngocele Successfully Treated by a One-Stage Surgical Procedure
Joseph A. Sabri

Branchiogenic Carcinoma
M. Stuart Strong and Seldon C. Sommers

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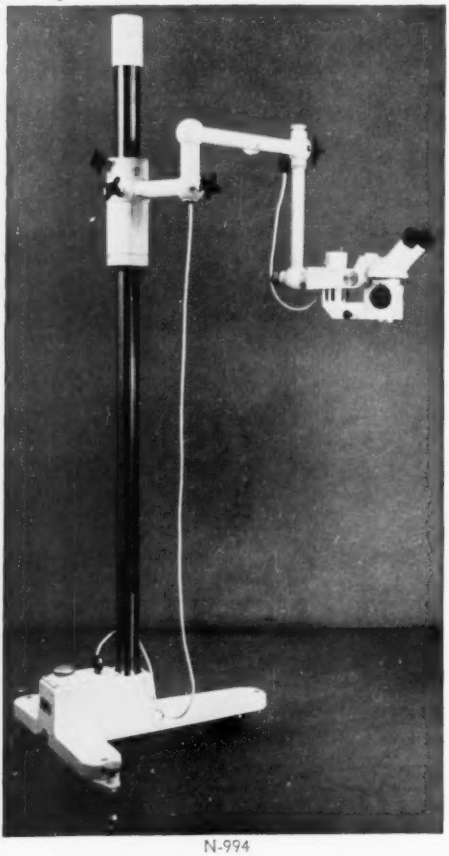

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TABLE OF CONTENTS

VOLUME 68 DECEMBER 1958 NUMBER 6

ORIGINAL ARTICLES

PAGE

Myocardial Functional Failure Occurring in Respiratory Obstruction
James F. Gardner, M.D., Rochester, N. Y. 659

Stapes Mobilization
Edward H. Campbell, M.D., Philadelphia 663

Growth of the Nose and the Septorhinoplastic Problem in Youth
Joseph G. Gilbert, M.D., Roslyn Heights, N. Y., and Samuel Segal Jr., M.D., Springfield, Mass. 673

Peristapedial Pathology and Otitic Reinfection
George Kelemen, M.D., Boston 683

Recruitment Measured by Automatic Audiometry
Bernard A. Landes, Ph.D., Lubbock, Texas 685

Subcuteneous Emphysema
Burton R. Andersen, M.D., Minneapolis 697

Nasal Sinus Disease
Henry M. Goodyear, M.D., Cincinnati 700

Intranasal Chromophobe Adenocarcinoma
Lawrence J. Lawson, M.D., Evanston, Ill. 704

Associated Carcinoma of Larynx and Lung
Alan S. Rubenstein, M.D.; James Graham, M.D., and Neale Barnes, M.D., Springfield, Ill. 710

Cerebral Mucormycosis
Howard W. Smith, M.D., and John A. Kirchner, M.D., New Haven, Conn. 715

Tracheostomy
John M. Loré Jr., M.D., New York 727

Secretory Otitis Media in Children
Pierce W. Theobald, M.D., Chicago 737

Secretory Otitis Media and Nasal Allergy
Don J. Weekes, M.D., Boston 748

The Education of a Young Deaf Child Begins with His Parents in His Home
Mrs. Spencer Tracy, Los Angeles 752

AUDIOLOGY SECTION

A Rapid and Simple Technique for the Detection of Nonorganic Hearing Loss
Dean A. Harris, M.S., Dallas, Texas 758

CASE REPORTS

Combined Internal-External Laryngocele Successfully Treated by a One-Stage Surgical Procedure
Joseph A. Sabri, M.D., Washington, D. C. 761

Branchiogenic Carcinoma
M. Stuart Strong, M.D., and Seldon C. Sommers, M.D., Boston 764

SOCIETY TRANSACTIONS

Chicago Laryngological and Otological Society 770

REGULAR DEPARTMENTS

Abstracts from Current Literature 778

Books 786

Directory of Otolaryngological Societies 787

A. M. A.
ARCHIVES OF OTOLARYNGOLOGY

VOLUME 68 DECEMBER 1958 NUMBER 6

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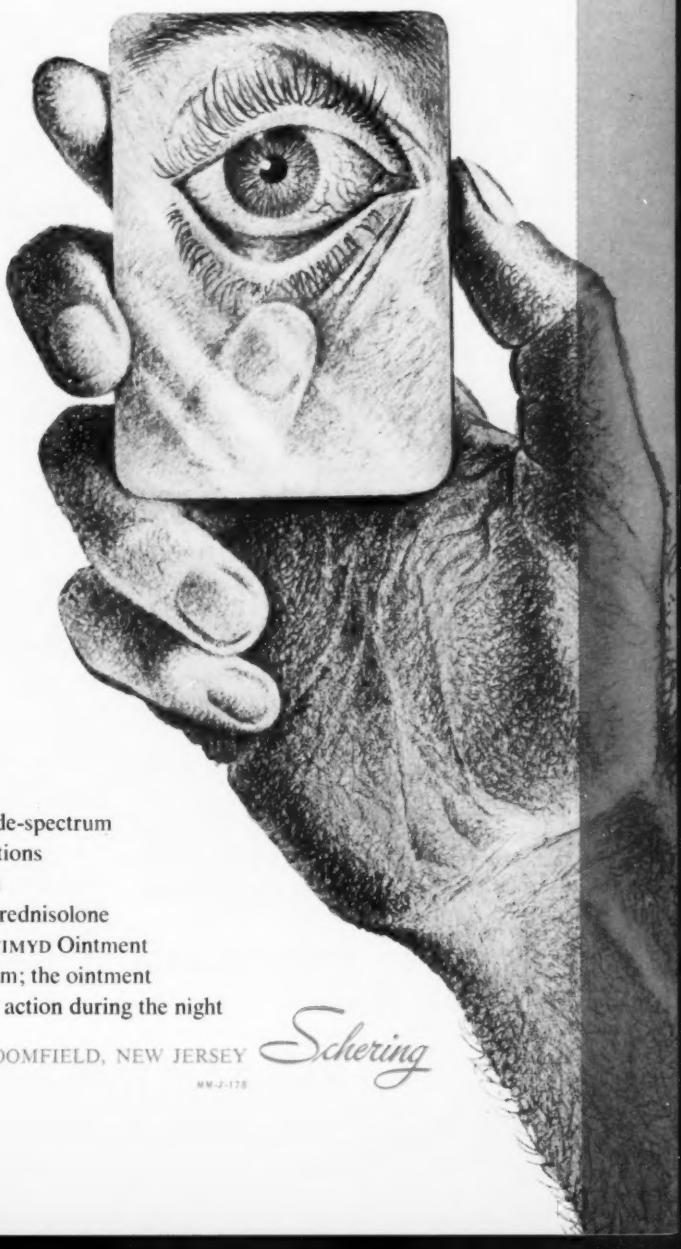

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Myocardial Functional Failure Occurring in Respiratory Obstruction

JAMES F. GARDNER, M.D., Rochester, N. Y.

Sudden severe respiratory obstruction always demands quick decisive action of the attending physician. The experienced laryngologist has the mechanism of tracheotomy so well rehearsed that he can perform the operation with a minimum of delay. Usually, the prompt relief of the respiratory obstruction by this procedure is all that is required to place the patient on the road to recovery. Occasionally, however, an extreme case is seen in which the anoxia has resulted in grave cardiac weakness and irregularity. When cardiac complications have already occurred, the execution of simple procedures to relieve the airway alone may be inadequate to assure survival of the patient. Cardiac arrest as a complication of surgical anesthesia has been thoroughly reported by many authors in recent years.[1-5] In the many papers discussing this problem, the necessity of avoiding anoxia is repeatedly stressed, for anoxia is considered the primary predisposing factor in cardiac arrest. The physiology involved in death from asphyxia seemed to basically parallel so-called "cardiac arrest" occurring in the operating room. Recent experience indicated that patients dying of sudden asphyxia not only do not die as a result of irreversible damage to medullary centers, but are as capable of resuscitation as are patients whose hearts fail on the operating table. A review of the basic biochemistry and physiology of the heart seems to establish a sound basis for definitive treatment of severe asphyxia. After a review of these fundamental data, several cases will be presented for analysis.

Submitted for publication April 3, 1958.

Presented as a candidate's thesis in partial fulfillment of the requirements for admission to the American Laryngological, Rhinological and Otological Society, Inc.

Basic Considerations

Some of the accepted facts relating respiratory and cardiac functions must be presented here, although it is not intended to discuss cardiac physiology in an authoritative manner.

Regarding utilization of oxygen by the heart, we can summarize the admittedly very complex biochemical reactions that result in the liberation of the energy required for contraction of the cardiac muscle by stating that the myocardium normally utilizes lactic acid for fuel. Lactic acid utilization by the heart corresponds to the work done by the heart. In this respect, the oxidation of lactic acid occupies a more important place in cardiac than in skeletal muscle contraction. In the latter, lactic acid is oxidized to resynthesize glycogen, the breakdown of which supplies the energy for contraction of the skeletal muscle. It is believed that glucose is used by the heart mainly to restore depleted glycogen stores, and glycogen is pictured as an emergency fuel used under extraordinary conditions. Glycogen stores

have been found to be rapidly depleted when epinephrine is administered or when oxygen is deprived. Apparently, then, lactic acid is the fuel under conditions of normal oxidation, but the stored glycogen is the only source of energy available to the heart when the oxygen supply is cut off. Utilizing the energy of glycogen breakdown, the mammalian heart can continue functioning for only 5 to 10 minutes after oxygen is deprived.[6]

Increase in CO_2 and pH such as occurs in asphyxia has been found to decrease the activity of the conductive or specific tissue of the heart. Irregularity and weakness of the beat progress to complete heart block and cardiac standstill as conduction is depressed. The heart muscle itself is highly sensitive to changes in the pH and has a much lower buffering power than has skeletal muscle. It may be for this reason also that the ability of cardiac muscle to function minus oxygen is only one-fifth as great as the ability of skeletal muscle.[6]

The common myocardium is especially vulnerable not only with respect to function in anoxia but probably also with respect to actual survival. It is the only tissue in the body which must have added substrate to utilize oxygen in the Warburg apparatus. Even brain tissue is more durable in this respect.[4]

The specific tissue also is very durable with respect to anoxia considering survival but fails to function normally with changes in pH, as mentioned above. It is thought that this failure of normal function under conditions of anoxia and particularly acidosis is the basic cause of so-called vagovagal reflexes which may result in sudden cessation of the heart beat. The heart in anoxia and acidosis behaves with respect to sudden cardiac arrest in a fashion similar to a heart damaged by vascular dysfunction or inflammation, particularly rheumatic fever.[4]

The pulmonary or lesser circulation has been extensively studied in recent years in conjunction with cardiac function. These investigations are particularly interesting with reference to asphyxia because they throw light on the method of production of pulmonary edema in anoxia.[8-11] There is general agreement that the pulmonary arterial pressure rises under conditions of anoxia and that this pressure continues to rise for several minutes in the mammal after the peripheral pressure has begun to fall. This fall in peripheral pressure occurs as the heart beat slows and becomes irregular under the continuing severe anoxia. Agreement is not universal as to whether the initial rise in pulmonary pressure is a result of a pulmonary reflex vascular vasoconstriction, or the result of the general increase in cardiac output [12] which occurs in slighter degrees of anoxia. The continued rise in the pulmonary pressure is attributed by all investigators to the ultimate failure of the left side of the heart. It is at this terminal point that pulmonary edema occurs, apparently entirely the result of left-sided failure.

In confirmation of this, one investigator [8] kept guinea pigs in a hypoxic environment until gasping movements occurred and then killed the animals before there was evidence of cardiac irregularity as indicated by ECG. The animals were guillotined at this point, and immediate analysis was performed without finding evidence of pulmonary edema. Guinea pigs allowed to die in hypoxia showed marked pulmonary edema on analysis.

Report of Cases

Patients not infrequently suffer bouts of acute asphyxia and come under the care of the physician in extremely critical conditions. The following three cases are presented in brief.

CASE 1.—An 8-year-old boy had had an upper respiratory infection with laryngitis for three days when he suddenly suffered a severe bout of obstruction at dinner. He was taken to the pediatrician's office, where he seemed to be breathing quite freely, only to suffer a second episode of respiratory obstruction, severe enough to cause loss of consciousness. He recovered consciousness about 30 minutes later, as he was admitted to the hospital. At this time, he was slightly cyanotic, was breathing with slight retraction, and his lungs were filled with coarse and fine rales. A foreign body

was diagnosed, an emergency tracheotomy and bronchoscopy were performed. His condition rapidly deteriorated; he lost consciousness, and failed to improve with the tracheotomy. Bronchoscopy through the tracheotomy revealed marked pulmonary edema but no foreign body. The bronchoscope was withdrawn, and an endotracheal tube inserted just as the heart stopped. Thoracotomy was done, and cardiac massage started within two minutes, with the simultaneous administration of oxygen under pressure. After 15 minutes, the heart resumed normal rhythm, and his color for the first time became normal. The pulmonary edema cleared rapidly. During closure of the chest, the heart stopped again. Massage again restored the beat only to have the heart stop a third time. Four times in all, the beat suddenly stopped, and the last time it could not be restored. Total time elapsing was four hours. Fibrillation occurred late and was controlled at first with KCl and later with the defibrillator. Epinephrine and procaine and calcium chloride were given repeatedly to increase muscle tone. Lanatoside C (Cedilanid) and atropine were also given.

CASE 2.—An 18-month-old boy was admitted to the hospital with severe croup. An endotracheal tube was passed at first with little relief of the obstructive symptoms, apparently because of failure to clear secretions from the tube. A short time later, a bronchoscope was passed, and oxygen administration was begun under pressure through the scope. As the bronchoscopy was being done, all respiratory movement ceased, and heart sounds were absent on auscultation. Within two minutes, a thoracotomy was done. The heart was seen to be in standstill, but as the ribs were forcibly spread it began to beat. Auscultation revealed the lungs to be filled with coarse and fine rales. Rotating tourniquets were applied to the extremities, and digitalization with Cedelanid was begun as the chest closure was started. At one point during the closure, the heart beat slowed and became irregular again as a result of the oxygen pressure inadvertently dropping for 30 seconds. The lung fields cleared over a period of one hour. Tracheotomy was performed at this time just as the patient began to respond to stimuli. Six hours later, he responded to spoken voice. His postoperative course was uneventful.

CASE 3.—An 8-year-old boy was admitted in an unconscious state after accidentally hanging himself. He was noted to be cyanotic, and coarse and fine rales were heard throughout his lung fields. Subcutaneous emphysema was present over the anterior cervical region. An overhead portable x-ray revealed severe pulmonary congestion. An ECG was interpreted as showing sinus tachycardia and anoxia. He was digitalized with lanatoside C four hours after admission and responded to stimuli three hours later. Cyanosis disappeared 12 hours after admission, and he was fairly lucid in 15 hours. His recovery was uneventful.

Comment

Each of these cases represents a variation of the problem of sudden severe respiratory obstruction followed by secondary myocardial functional failure and severe pulmonary edema. Failure to appreciate the significance of the pulmonary findings in Case 1 resulted in avoidable delay in administering oxygen under pressure. Tracheotomy alone was useless and only placed an additional strain on the damaged heart. This shows that cardiac function will not improve unless oxygen can be delivered to the myocardium in adequate supply. When oxygen was delivered under pressure, manual massage restored the beat, and the cyanosis rapidly cleared. It was apparent that massage, with oxygen under pressure, was delivering more oxygen to the tissues than was the spontaneous heart beat prior to arrest without oxygen under pressure. Similarly, in Case 2 cardiac function rapidly deteriorated when the oxygen pressure fell momentarily.

While the first slowing and standstill of the heart occurring in Case 1 was undoubtedly the result of anoxia, the subsequent sudden arrest was apparently reflex in origin. This is an illustration of the recognized fact that sudden standstill is more likely to occur in a damaged heart.[4] Of the three cases, there is little doubt that this boy suffered the longest period of anoxia. Atropine was given to this patient after the first sudden arrest occurred, for the same reason that it is routinely used preoperatively: to minimize the danger of vagovagal reflexes. Although it is generally considered very efficacious in this respect, here it proved of little value, probably because of the extent of existing damage.

The action of digitalis and the other cardiac glucosides is thought to be mainly in improving the utilization of chemical energy by the myocardium. They exert no beneficial effects upon myocardial insufficiency due to anoxia, while the anoxia persists.[6]

They were given in the above cases in the hope that the heart would be strengthened as the anoxia was counteracted. It seemed to be of definite benefit in Cases 2 and 3. The use of cardiac glucosides, moreover, has been recommended by various authors dealing with cardiac arrest, particularly where there is accompanying pulmonary edema.[4]

In Case 3, the laryngologist was consulted because of the presence of rather marked subcutaneous cervical emphysema. The possibility of a prophylactic tracheotomy was being considered. The cardiac complications were noted and the idea of immediate tracheotomy rejected in favor of "wait and see" observation. It was recognized that the operation would place an additional strain on the damaged heart. Subsequently, a tracheotomy was unnecessary.

In cases such as we are considering, bronchoscopy is the method of choice in relieving the obstruction because it can be done with ease and speed. Attention can then be directed to the cardiac problems as in Case 2 and the tracheotomy deferred for a time to the advantage of the patient. If cardiac arrest occurs, the surgeon should be ready to enter the chest immediately and attempt to restore cardiac function. The time interval is of the greatest importance in successful cardiac massage. After four minutes elapses, it is probable that irreversible brain damage occurs. The possibility of restoring cardiac function after such damage has occurred is slight, as clinical experience shows, probably because the fragility of the myocardium approximates that of brain tissue. The problems of cardiac massage are beyond the scope of this paper, but every surgeon should recognize the need and the value of the procedure. It is the prompt application of basic principles that may be life-saving where the delayed efforts of the more experienced are fruitless.[7]

Summary

It is important to evaluate the cardiac status of patients in acute severe respiratory obstruction because pulmonary edema secondary to myocardial functional failure may be present. The administration of oxygen under pressure is essential, and this makes bronchoscopy the method of choice to relieve the obstruction. The basic principles of instituting cardiac massage should be understood by every laryngologist.

277 Alexander St. (7).

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Stapes Mobilization

Analysis of Results in Two Hundred Fifty Operations

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In the past four years the advances in the operative technique of stapes mobilization have been rapid and of great importance. With these changes of technique has come a steadily increasing percentage of good hearing results, starting with the 30% to 40% successful mobilizations reported by Rosen[1] in 1953 and increasing to the 70% to 80% successes indicated by recent reports of Rosen,[2] Goodhill[3] and Scheer.[4] It is difficult to evaluate the results recorded by some authors, as in some instances their successes are registered only by an "improvement of hearing" or a "significant gain" without indicating whether or not the improvement has been to the 30 db. level or better. A significant gain might mean only an improvement of hearing of 10 db. regardless of the level attained, while a gain of real significance would be the improvement to the bone-conduction level or better, whether or not this gain had reached the threshold of practical hearing. In estimating the results of a particular mobilization technique a success may be rightly claimed by the improvement of hearing to the level predicted by a study of the preoperative bone conduction regardless of whether or not this level was to the 30 db. line. As far as technique alone is concerned, such an improvement would indicate a greater success than the improvement of a patient from the 40 db. line to the 25 db. level when the improvement should have been 10 to 15 db. more to reach the predicted level.

The evolution of mobilization techniques has been through logical steps from the original method of Rosen,[1] which attempted mobilization at the stapes neck, through the method of pressure on the incus, various maneuvers at the stapes head, and finally to manipulations of various kinds to the stapes footplate. It appears that numerous operators, at more or less the same time, have reached the conclusion that more successful mobilization can be accomplished by manipulations of the footplate and taking

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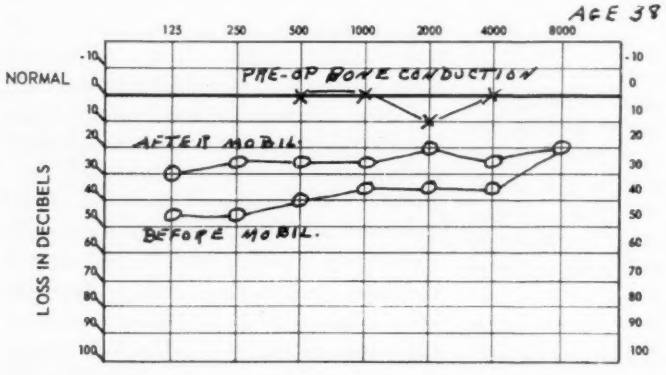

Fig. 1—Hearing before and after mobilization. Note improvement to above 30 db. line but not to preoperative bone-conduction level.

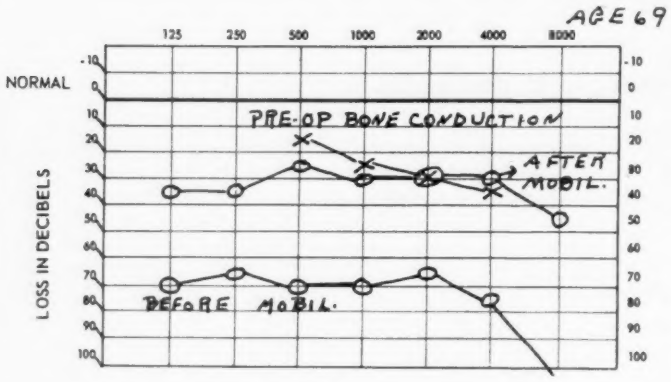

Fig. 2.—Hearing before and after mobilization. Note age of patient and improvement to preoperative bone-conduction level.

care to avoid dislocation of the incudostapedial joint and fractures of the crura. For more complete stapes mobilization and higher percentage of successful attempts there is now general agreement that manipulations at the footplate with avoidance of crural fractures and dislocations of the joint give the best chances for successful results.

Technique of Mobilization

In this presentation it is my intention to avoid any detailed description of surgical techniques. Recent publications have covered thoroughly the various details of techniques employed by different operators. It appears that each surgeon modifies previously described maneuvers to suit his own individual preferences and everyone reaches a systematic method of carrying out each step of the operation. A significant feature of the results reported by various operators is the steady increase of the percentage of successful mobilizations as their experience in this operative procedure has increased. While much of this higher percentage of successful attempts has resulted from the change of the activities to the footplate from the attacks on the incus and stapes head and neck, some of the greater success has undoubtedly resulted from the increased skill in the delicate manipulations, gained by much experience.

The surgical technique employed in this series of mobilizations has been variable. In none of them has an attempt been made to mobilize the stapes by the original method of Rosen—by force applied to the neck of the stapes. This method has not seemed as logical as force applied through the lenticular process of the incus or directly on the capitulum of the stapes. In the early cases, therefore, mobilization was attempted by the transincudal pressure method described by Goodhill[5] with at times use of pressure on the projecting edge of the stapes head. Analysis of the results obtained by these early and comparatively few attempts indicate that many failures occurred as the result of fracture of one or both crura or dislocation of the incudostapedial joint. Attempts at mobilization were therefore changed largely to manipulations at the footplate, and this method has been used in all the cases except the first 50.

Exposure of the incus and stapes has been produced as described by other operators—incision through the skin of the posterior auditory canal, elevation of the skin, deflection of the drum anteriorly, including the chorda tympani nerve when necessary, and removal of some of the annulus tympanicus. Palpation with the sharp curved pick or the blunt-ended mobilizer of Derlacki on the incus immediately overlying the stapes head is then made to determine three points: (*a*) the degree of fixation of the stapes footplate; (*b*) the looseness or

rigidity of the incudostapedial joint, and (*c*) the fragility or firmness of the crura.

Great care is taken to avoid pressure sufficient to produce fracture of the crura or dislocation of the joint. With the blunt-ended mobilizer pulsating pressure is then applied at the anterior end of the stapes footplate. If there is any suggestion of movement obtained, pressure is then made on the incus, keeping in mind the characteristics of the crura and joint ascertained by the original palpation. By a series of pressure applications alternating between various parts of the footplate and the incus or stapes head, free movement of the footplate is eventually obtained in the great majority of cases.

The completeness of the mobilization is determined by several observations: (*a*) the free mobility of the footplate both at the anterior end and the posterior end observed by direct magnified vision; (*b*) the detection of nystagmus occurring on light pressure on the incus; (*c*) the statement of the patient that a rotatory or up-and-down movement is experienced; (*d*) the characteristic feel of a mobilized footplate with the easy movement inward and the quick firm spring-back or rebound of a loosened footplate that still retains an intact annular ligament; (*e*) testing of the hearing both with the middle ear opened and after the drum and skin of the canal have been replaced.

The operative procedure is completed by applying a small strip of paraffin mesh gauze soaked in Neosporin ointment (antibiotic ointment containing polymyxin B, bacitracin, and neomycin), which rests lightly on the drum and against the walls of the auditory canal. This packing is removed on the day following the operation, and on removal there is brought with it blood clots that have accumulated in the canal and would have accumulated on the drum. This obviates the necessity of removing blood clots from the drum and deep part of the auditory canal at subsequent visits. Furthermore, it leaves the canal open, so that, if the mobilization has been successful, the hearing is very frequently improved immediately. In most of the cases a detailed audiogram is taken on the first or second day following the operation after the packing has been removed. The second test is usually done on the 7th to 10th day after operation and the third test between the third and fourth week.

Analysis of Results in 250 Mobilization Cases

The statistics in this analysis are derived from the study of 250 consecutive mobilization operations performed between September, 1956, and February, 1958. Twelve of these operations were attempts at remobilization three or more months after the original operation. The results mentioned have been obtained by tests made from 1 month to 16 months after operation. The recording of these results is based on the following system averaging the decibel loss for the three speech frequencies only.

Plus 4 (4+) =Hearing improved to within 7 db. of the normal line.
Plus 3 (3+) =Hearing improved to the 30 db. level or better.
Plus 2 (2+) =Hearing improved at least 15 db. and to the 40 db. level or better.
Plus 1 (1+) =Hearing improved 10 db. or more but not to the 40 db. level.
ZERO (0) =No change of hearing over preoperative level.
Minus 1 (1—) =Hearing less than 10 db. worse than the preoperative level.
Minus 2 (2—) =Hearing 10 to 15 db. worse than preoperative level.
Minus 3 (3—) =Hearing more than 15 db. worse than preoperative level.

Plus 4 Cases.—In 11, or 4.4% of the cases, the improvement has reached to practically the normal line. All of these were of the "A" suitability type, and mobilization was accomplished by light pressure on the incus in seven instances and by a combination of light pressure on the anterior part of the footplate and on the incus in four cases. Four of these patients had 45 to 50 db. loss before operation, and the remaining seven patients had a preoperative hearing loss of 35 to 45 db.

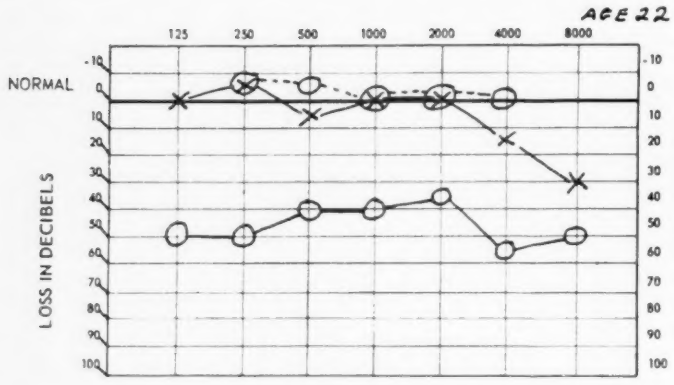

Fig. 3.—Improvement of hearing to normal after stapes mobilization. Solid line with circles indicates hearing before operation; broken line with circles, preoperative bone conduction; broken line with crosses, hearing after operation.

Plus 3 Cases.—One hundred twenty additional patients, or 48% of the two hundred fifty cases, received a plus 3 result, the hearing in all reaching a level of 30 db. or better. Adding this 48% to 4.4% receiving a plus 4 result gives 52.4% of the cases operated on which reached a practical hearing level. The great majority of these cases were of the "A" suitability type but some of them were "B" suitability cases, and a few of the "C" classification.

Plus 2 Cases.—Fifteen patients, or 6%, received an improvement which was designated plus 2, as they obtained 15 db. or more improvement that brought their hearing to a level between 30 and 40 db. loss. Seven of these fifteen received an improvement to the level predicted on the basis of their preoperative bone conduction and these were considered successful. The other eight patients obtained an early improvement well above the 30 db. level but subsequently lost part of this improvement to below the 30 db. level. These eight cases were considered failures. This 6% of the operated cases represents a significant gain of hearing but not complete rehabilitation. Six of the patients had a preoperative air-conduction loss of 60 to 70 db., and the improvement to the plus 2 level after operation has given them great satisfaction.

Plus 1 Cases.—These consist of those cases which received 10 db. or more improvement but which did not reach the 40 db. level. Some of these are patients who received a satisfactory early improvement but subsequently lost nearly all of the early gain. Some of these were remobilized, as will be recorded below. There were 26 patients, or 10.4%, in this group.

Zero (0) Cases.—These cases include those that received no gain or loss of hearing following operation and those that were improved satisfactorily for a short time but later lost all the improvement. Fifty-seven, or 22.8% of the 250 mobilizations, were of this type. In some of these an attempt at remobilization was done and in other cases a fenestration was performed as recorded below.

Minus 1 Cases.—In 13 patients, or 5.2% of the 250 cases, the final hearing result was a loss of 10 db. or less over the preoperative level. In most of these cases the patient was not aware of any increased hearing loss, and six of them had very satisfactory results after the fenestration operations. The remaining seven have not, thus far, had the fenestration operation.

Minus 2 Cases.—In six cases, or 2.4%, the hearing after mobilization was worse by 10 to 15 db., and again two of these patients received a satisfactory result after fenestration.

Minus 3 Cases.—In two, or .08%, the hearing became worse by 15 db. or more after operation. In both of these cases the further loss of hearing was distinctly noticeable to the patients and there was no difficulty in inducing them to have the

fenestration operation. In both instances a plus 3 result was obtained by the fenestration.

In all of the minus cases a satisfactory explanation of the failure was obtainable. In 18 of the 21 minus cases either the stapes crura were fractured or the incudostapedial joint dislocated. Two cases became slightly worse because mobilization was not accomplished and a small perforation of the drum occurred which thus far has failed to heal. An attempt at remobilization with a myringoplasty is planned for both of these patients. In one patient there was apparently some damage to the membranous labyrinth, as the postoperative bone conduction was reduced as well as the air conduction.

Of the 250 mobilized patients 16% (40 patients) obtained immediate good improvement but later lost part or all of the improved hearing. From a study of these cases with frequent postoperative testing it seemed evident that most of these lost the initially improved hearing because of refixation of the stapes footplate. In some, however, the cause could not be ascertained, and it is suspected that the causative factor in these was the closure of a fenestrated footplate.

A second attempt at mobilization on the same ear was made on 12 of the patients when the first mobilization was not entirely successful. In some of these a slight to moderate improvement of hearing was obtained after the first mobilization but not the amount of improvement that was expected. It seemed apparent in these cases that incomplete mobilization of the footplate had occurred, and the revision was attempted to obtain more complete loosening of the footplate. Four of these revisions were very successful, giving a plus 3 result, and three additional ones gave a plus 2 result. Three of the four revision failures were later fenestrated with an excellent result in three of them.

In order to determine the contributing factors to such unsuccessful cases it is necessary to make postoperative tests at frequent intervals. If possible the first audiometric test is made the first or second day after operation, when the patient is discharged from the hospital and the small strip of gauze packing has been removed. In spite of the traumatic swelling of the drum, blood clots in the canal, and blood in the middle ear, the audiogram a day or two after operation will show a considerable improvement of hearing in those cases that have been satisfactorily mobilized. This early improvement usually is most prominent at the lower frequencies, and there is little or none at the 2000 frequency. Subsequent tests during the next three weeks will show a further improvement to the lower frequencies and a change for the better at the 2000 and higher levels. In the successful cases the hearing reaches its best level at the high frequencies during the third and fourth week after operation.

When audiograms are taken at five- to eight-day intervals during the first month after operation, much can be learned regarding the outcome of the case. A steady improvement of hearing during the first two or three weeks is a very good indication that the eventual results will be good. Some of the patients show a satisfactory improvement a day or two after operation but suddenly lose part or all of this improvement from one to three weeks later. In those cases in which this happens the hearing recedes to the preoperative level or near this level within five weeks after operation in the great majority of cases. Occasionally there is a more gradual loss of the early improvement over a period of three to four months, but in the great majority of cases the hearing will become worse within a month after operation if it is going to do so at all.

In this series of 250 mobilizations there were 16% (40 cases) that obtained good improvement of hearing immediately after operation but later lost part or all of the improvement. Some of these obtained a lasting improvement after a revision. A

favorable time for revision was considered anytime after three months from the time of the original mobilization.

Results Obtained in 250 Mobilization Operations

In evaluating the results obtained in this series the cases have not, at this time, been separated into the usual three classes of suitability for operation. It has been observed, as others have recorded, that the highest percentage of good results is obtained in the "A" suitability group. It has further been observed in these cases that anticipated and predicted results have been observed in the "B" and "C" group in as high a percentage as in the "A" group. This would appear to indicate that mobilization can be accomplished in the "B" and "C" groups in as high a percentage as in the ideally suitable patient. It has also been observed that the older patients may mobilize as easily as or perhaps more easily than the young patients. In this series there have been 30 patients past 55 years of age where mobilization has been attempted, and 46.6% of them have had completely successful results. The preoperative hearing loss in these has, on the whole, been greater than in the younger patients with presumably a firmer fixation of the stapes. It would appear that the otosclerotic bone producing the fixation in the older patients has become brittle and therefore more easily broken.

In this series of 250 mobilizations the hearing has been improved to the 30 db. level or better in 52.4%. An additional 6.4% obtained a significant gain (15 db. or more) and the majority of these reached the preoperative prediction level. In dividing this series into two groups of 100 cases each and one group of 50, some significant features have been observed. In the first 100 cases 42% obtained an improvement above the 30 db. level (plus 3 and plus 4 cases); an additional 5% received a significant gain, and 17% made 8 to 15 db. worse. In the second 100 cases 52% obtained improvement above the 30 db. level; 6% received significant gain of hearing, and 10% were made 8 to 15 db. worse. In the last 50 cases 70% were improved above the 30 db. level; 10% additional received significant gain in hearing, and 4% were made worse by less than 10 db.

These statistics showing the improvement of the percentage of successful mobilizations and the reduction of cases in which the hearing has been made worse reflect the trend shown by all operators. The chief factor which is responsible for the greater success in the more recent cases is probably the change in the manipulations to the footplate from the earlier attempts at the incus and the head and neck of the stapes. In the majority of these cases in which one or both crura have been fractured or the incudostapedial joint disarticulated the hearing will become worse after operation. In such cases, even if the footplate has been mobilized, there appears to be little or no improvement of hearing and at times the hearing may become worse. It has been observed that in those cases with a preoperative loss of hearing of 30 to 35 db. the fracture of the crura or dislocation of the joint will very commonly result in a loss of hearing after operation to a level greater than the preoperative level.

It has been mentioned that the mobilization operation is indicated in those cases of relatively mild preoperative hearing loss such as 30 to 35 db. loss, but it must be kept in mind that fracture of the crura or dislocation of the incudostapedial joint can make the hearing in these cases distinctly worse. It has also been observed that these cases of moderate preoperative loss are not necessarily easier to mobilize. In fact I have seen cases of 30 db. preoperative loss who were more difficult to mobilize than those patients 60 or more years of age with a preoperative loss of 60 db. Extreme care, therefore, is required in those patients of mild to moderate hearing loss to avoid the common accidents of crural fracture and joint dislocation. These patients with the mild preoperative loss are far from happy when the hearing is worse after operation.

Unsuccessful Mobilization Cases

It has been the common practice to consider successful those patients who have maintained a satisfactory improvement of hearing for a month or more after operation. From the study of cases which have been mobilized for five years or more it does appear that nearly all patients who have maintained the initial improvement for a month after operation will continue to retain this improvement. In my own series I have observed the earliest cases only 16 months, but these cases which were successful have maintained the hearing at their best levels during this time except in very few instances. What will happen to the hearing in these patients in subsequent years can only be known by continued observation. It would seem important to continue postoperative observation and testing of these patients for an indefinite time rather than to consider them successful indefinitely after a few weeks' or months' observation and permit them to get entirely out of the operators' hands.

Attempts at stapes mobilization may be unsuccessful from several causes. With the more recent technique of manipulation at the footplate under high magnification, failures have been reduced considerably, partly because of the fewer crural fractures and joint dislocations and partly because of more accurate instrumentation at the footplate itself.

At times failure will result from incomplete mobilization. To detect these cases it is important that observations at the time of operation be exact, the recording of the operative findings be accurate, and frequent postoperative observation and testing be made. Most of such cases are amenable to revision, at which time more aggressive manipulations may produce a complete success. I have performed revision on 12 cases in which an apparent incomplete mobilization resulted at the first operation. Four of these revisions were entirely successful and three additional ones gave significant gains of hearing.

In those cases of advanced diffuse otosclerotic stapes fixation (approximately 10%) mobilization appears impossible or at least impractical. Failures in an additional substantial number will occur from fracture of the crura and dislocation of the incudostapedial joint. Other failures may occur from anomalies of the conduction mechanism and adhesive formations in the middle ear. All such failures are not suitable for attempts at remobilization, and the only prospects for satisfactory hearing improvement is with the fenestration operation.

Fenestration After Mobilization

Up to the present time there have been no statistics showing the results in any substantial number of fenestration operations following unsuccessful mobilizations. I wish here to make a preliminary incomplete report on 43 fenestrations following unsuccessful mobilization attempts. All of these have been done within the past 13 months, and the results thus far obtained cannot be considered final, but certain observations have been made that appear to have some significance.

A few years ago, when satisfactory results from mobilization became firmly substantiated and received considerable publicity, great interest was aroused in deafened persons. It has been amazing to hear of and to see the great number of otosclerotics who wish to have the mobilization operation who have not, in the past, been interested in the fenestration operation. The handling of these patients has at times been difficult. For some of us who have been actively engaged in fenestration surgery, it has not been easy to consider giving up an operation in which 80% or more good results can be obtained to perform an operation that has appeared to be successful in 50% or less of cases.

It has been my policy, in interviewing these patients, after having confirmed by testing their suitability for operation, to suggest the mobilization operation as a trial

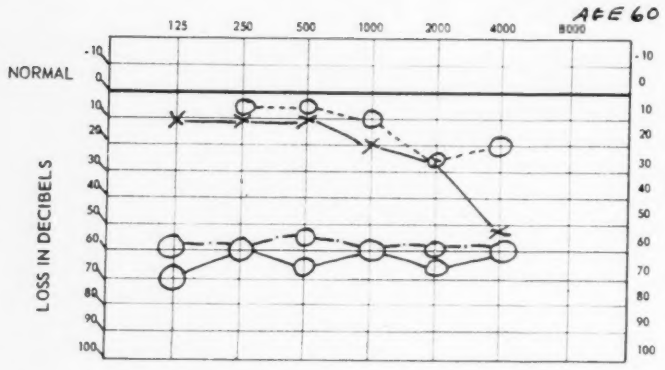

Fig. 4.—Successful fenestration after mobilization failure. Note age of patient. Solid line with circles indicates hearing before mobilization; dot-dash line with circles, hearing after mobilization; broken line with circles, preoperative bone conduction; broken line with crosses, hearing after fenestration.

or a procedure preliminary to the fenestration operation. I have strongly urged them to have the fenestration three months to a year after the mobilization, if the mobilization has not been successful. By presenting to them the main features of both operations, prospects of each, postoperative reactions, etc., I have been able to induce a rather high percentage to have the fenestration operation when mobilization has been a failure. At the present time with a successful result in 60% or more of the mobilizations and 80% or more fenestration successes with the mobilization failures, extremely good prospects can be offered the otosclerotic patients.

In this series of 250 mobilization cases failure to obtain satisfactory mobilization occurred in 112 cases, or 44.8%. The fenestration operation was performed on 43 of these 112 failures. The results are, as yet, not entirely acceptable, as nearly all of them have been done less than a year. However, the early results in those 43 cases have been as good and the percentage of good results as high as in all of the fenestrations I have done previously.

At the present time 79% of these 43 fenestrations have been entirely successful, improving the hearing above the 30 db. level. An additional 12% have had substantial improvement to within 10 db. of the 30 db. level. In half of these 12% the hearing has been improved to the level that could be expected when the preoperative bone conduction is considered. A gratifying feature has been the very successful results obtained in those patients in whom the hearing was made worse by the mobilization operation. In two of these mobilization failures the hearing was made worse by more than 15 db. Both of these obtained

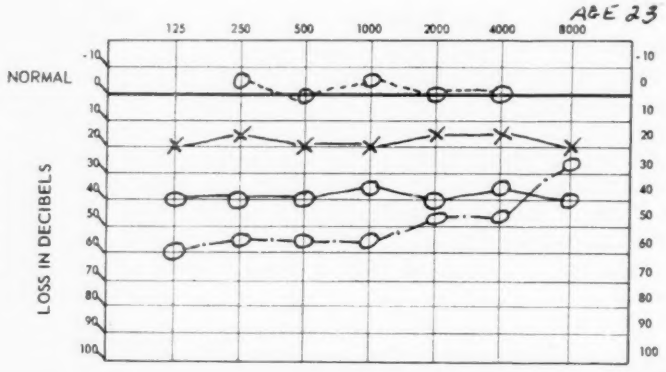

Fig. 5.—Successful fenestration after mobilization failure. Note the minus 3 result after mobilization. Solid line with circles indicates hearing before mobilization; dot-dash line with circles, hearing after mobilization; broken line with circles, preoperative bone conduction; broken line with crosses, hearing after fenestration.

improvement above the 30 db. level with the fenestration operation. In eight other mobilization failures, with the hearing 8 to 15 db. worse after the mobilization, the fenestration produced a plus 3 result. It is anticipated that others of these mobilization failures will have the fenestration operation later.

In these fenestration operations there have been no difficulties encountered which could be attributed to the previous mobilization procedure. Considerable adhesions have been found in the middle ear in some of the cases, but there has been no defect in the skin flap as a result of the incisions made in performing the mobilizations. The postoperative reactions have been identical with the fenestrations previously performed.

Complications

Complications in this series of cases have been few and mostly of a minor nature. The chief one encountered has been prolonged postoperative dizziness or sense of unbalance. This has occurred more frequently since manipulations have been made chiefly at the footplate. Occasionally the postoperative vertigo has been severer than that observed after the fenestration operation. In the great majority of instances the balance is fully adjusted in three to six days and the patient can resume his full activities.

A rather common complication is perforation of the tympanic membrane. In nearly all cases this will heal spontaneously within two or three weeks. In two instances in this series rather large marginal perforations have persisted. One of these was completely healed with an excellent hearing result by a combination fenestration and myringoplasty. In another patient mobilization was successfully accomplished, but a large marginal perforation remains. A myringoplasty is planned for this patient in the near future.

In one patient there has apparently been some damage to the inner ear mechanism, as the air-conduction hearing has been made worse and the bone conduction has been reduced over the preoperative level. No adequate explanation is apparent as mobilization was not accomplished.

There have been no instances of facial paralysis in this series and no primary infections of the ear canal or the middle ear. Two patients developed middle-ear infections two and three weeks after mobilization. In both cases perforations had occurred in the drums, and acute nasal infections later had produced contamination in the middle ear. Both of these middle-ear infections cleared up within a month, and the perforations healed spontaneously. Both patients have had excellent hearing results.

Summary and Conclusions

An analysis of results obtained in 250 stapes mobilization operations is presented.

The technique employed, in the great majority of the cases, has been a pulsating pressure applied alternately to various parts of the stapes footplate and the incus and stapes head.

The results have been judged largely on the basis of improvement of hearing to the 30 db. level or better and the improvement to or near the level predicted on the basis of the preoperative bone conduction.

In 11, or 4.4% of the cases, the improvement of hearing has reached to practically the normal line (an average of less than 7 db. loss to the speech frequencies).

In 120 additional patients, or 48% of the 250 cases, the hearing was improved to the 30 db. level or better. A practical serviceable hearing level was, therefore, obtained in 52.4% of the cases.

Fifteen patients, or 6%, received an improvement of 15 db. or more, and in seven of these the hearing was improved to the preoperative prediction level.

In 8.4% of the cases the hearing was made worse by the operation. Most of these were worse by less than 10 db. The factors concerned in the greater loss of hearing after operation appeared to be

fractures of the crura and dislocation of the incudostapedial joint in nearly all the cases.

A mobilization revision was performed on 12 of the patients; four of the revisions were entirely successful and three additional ones were partially successful.

The analysis indicated that mobilization could be accomplished in as high a percentage in the "B" and "C" suitability groups as in the "A" or ideally suitable patient.

It was further observed that mobilization was as successful in the patients over 55 years of age as it was in the group 20 to 30 years of age.

Mobilization was unsuccessful for the following reasons: (*a*) incomplete or partial loosening of the footplate; (*b*) fractures of one or both crura; (*c*) dislocation of the incudostapedial joint; (*d*) in those cases of advanced diffuse otosclerotic stapes fixation; (*e*) anomalies of the conduction mechanism; (*f*) adhesive formations in the middle ear.

The fenestration operation was performed on 43 of the 112 mobilization failures. Of these 79% have been entirely successful and an additional 12% have been partially successful.

Complications have been of a minor nature, consisting of prolonged dizziness or sense of unbalance, occasional perforations of the tympanic membrane, and secondary middle-ear infections in two cases.

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Growth of the Nose and the Septorhinoplastic Problem in Youth

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The management of the recently acquired or old deviated obstructing septums in children is always difficult. The desire to be surgically conservative with the "growing nose" runs into the practical problem of attempting to relieve any nasal respiratory obstruction caused by the deviated septum. This is further complicated, on the one hand, by the desire to correct any external nasal deformity that might be present and, on the other, by the possibility of its occurrence as the result of surgery on the septum.

In order to better understand the reasons for or against surgery and the type of surgery to be performed in children with deviated septums, with or without external nasal deformities, it is necessary to understand the following:

1. The embryology and growth of the component parts of the nose.

2. The relative importance of each of these components in the *maintenance* and in the *continuous growth projection* of the *nasal profile* during the growth period.

Embryology and Growth of the Nose

The Bony Vault

The bony vault consists of the nasal bones, nasal processes of the maxillae, and the nasal processes of the frontal bone. The nasal bones and nasal processes of the frontal bone arise from the embryologic frontonasal process, while the maxillae develop from the first (mandibular) arches.

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1. Nasal Bones.—A. Embryology: The nasal bones arise as membranous structures lying on the surface of the cartilaginous nasal capsule. At about the fifth week of fetal life, single centers of ossification appear in the membranous tissue. While the ossification of the nasal bones proceeds, the underlying cartilage of the nasal capsule, which acts as a framework around which the nasal bones grow, is absorbed.[1,2,3] However, based on the well-known fact that the upper lateral cartilages are attached to the undersurface of the nasal bones rather than an end-to-end attachment, the above statement should be modified to read "absorption of the underlying cartilage of the nasal capsule is complete except for a small rim of cartilage which remains under the caudal margin of the nasal bones." The nasal bones continue to grow to age 14-17 years.

That the nasal bones can develop properly, unequally, poorly, or not at all, may be readily understood from the above-described embryology. Where a total absence of the nasal bones occurs, the nasal processes of the maxillae not only grow cephalad but also project mesially and fill in the vacancy created by the absence of the nasal bones.

What effect have the nasal bones on the development of the cartilaginous nasal vault?

In view of the fact that the upper lateral cartilages are attached to the undersurface of the nasal bones, any growth of the nasal bones in either a ventral or a caudal direction carries the upper laterals along with them. In turn, any change in position of the latter is reflected to the underlying connected ventral margin of the cartilaginous septum.

As will be shown in the discussion on the clinical evaluation of the embryology of the nasal bones, this is of minor importance in the final determination of the growth of the cartilaginous vault and septum.

B. Clinical Evaluation of the Embryology:

1. In spite of fractures sustained by nasal bones in early life with resultant saddles in the bony vault, the projection of the cartilaginous vault may grow to normal heights. This fact points up the relatively unimportant influence the nasal bones have over the projection of the cartilaginous vault and its underlying septum.

2. In view of the fact that there are only single centers of ossification for each of the nasal bones, injury to such a center can cause abnormalities in the anatomical growth of the nasal bones.

3. The earlier in life trauma occurs to or operation is performed on a nasal bone, the more damage to growth may ensue.

4. Regardless of the exact location of the ossification center in the nasal bones, injury to them could hardly be avoided during rhinoplasty or open reduction of fractures in children. This is likely, considering the amount of elevation of skin and periosteum that is required during those procedures, and the small size of the nasal bones. Therefore, open reduction of fractured nasal bones or rhinoplastic procedures on the bony vault should not be done before maturity of the nasal bones, i. e., 14-17 years. Closed reduction of nasal bone fractures in children are as a rule most successful.

II. Maxillae.—A. Embryology: The maxilla first starts out as a membranous structure. At the sixth to seventh week of fetal life, ossification starts from a center above the canine tooth germ and spreads in all directions. At about the fourth month of fetal life, the maxilla which is ectal to the cartilagenous nasal capsule invades this capsule and incorporates the adjacent lateral wall of the capsule. The maxilla thereby participates in the formation of the lateral walls of the nose as well as being the recipient of the nasal mucous membrane pouch which subsequently becomes the maxillary sinus.

At birth, the average forward position (ventrally) of the maxilla is about 7 mm. At the age of 6 years, it has pushed itself forward to about 27-28 mm., an increase of 21 mm. (better than 4/5 in.). From then on, however, the rate of growth is at a snail's pace, in that from the 7th to the 15th year of life it grows ventrally only about 4 mm. (less than 1/5 in.).

The nasal processes of the maxillae normally help make up the lateral walls of the nasal bony vault. Therefore, the maxillae contribute to the width as well as to the profile projection of the bony vault. This contribution to the projection of the bony vault takes place principally during the first six years of life, as was indicated above.

As previously stated, if the nasal bones are absent the nasal processes of the maxillae will meet in the midline and make up for the undeveloped nasal bones. This also indicates their ability to contribute to the bony nasal profile.

B. Clinical Evaluation of the Embryology:

1. Fractures of the nasal processes of the maxillae before 6 years of age can severely interfere with the growth and projection of the nasal bony vault. After 6 years of age, they could also interfere with the projection of the bony vault, but to a less degree.

2. In unilateral cleft lips, on the side of the cleft there is "usually underdevelopment of the maxilla" as well as "asymmetry of the nasal bones," according to Holdsworth.[4] In our opinion, however, the latter results from poor growth of the nasal process of the maxilla on the side of the cleft rather than actual deformity of the nasal bone per se, as is implied in the above statement. This opinion is supported by Schaeffer's[1] statement that "developmental processes go hand in hand and that when a certain time is reached a structure will normally have attained proportions for that period regardless of related parts."

3. In repair of harelip and cleft nares, if the periosteum over the canine tooth area is elevated, and this is frequently done, the future growth of the maxillae and the nasal process of the maxillae will be further compromised. In this manner the projection of the bony vault is further interfered with.

III. Nasal Processes of the Frontal Bone. Single centers of ossification appear in the frontal bones between the sixth and seventh weeks of fetal life. Bony processes then appear in the roof of the nose in front of the cribriform plate and separate the nasal cavity from the frontal sinus. From the center of the nasal process of the frontal bone, the frontal spine, arising from its own centers of ossification, projects downward and forward into the nasal septum between the nasal crest of the nasal bones and the perpendicular lamina of the ethmoid, articulating with both, forming a miter joint —probably the strongest of all joints.

With the marked increase in the development of the frontal lobe in higher animals, the frontal bone and the nasal processes of the frontal have taken a more ventrocaudal position, with the result that the profile projection of the nose is most marked in higher animals and especially in the human.

The Cartilaginous Vault

The cartilaginous vault is composed of the upper and lower lateral cartilages.

Lateral Walls.—A. Embryology: The lateral walls of the nasal pyramid in fetal life are made up of one continuous sheath of cartilage. Chondrification of this vault occurs during the third month of fetal life independently of the septum. Eventually, however, the lateral cartilaginous walls join the cartilaginous nasal septum. At the sixth month of fetal life an ingrowth of connective tissue from the dorsal margins of the lateral wall, at its juncture with the "pyriform" margin of the maxilla, divides the single cartilaginous vault into upper and lower lateral cartilages and membranous septum.

How does the architecture of the lateral cartilages aid in the growth of the profile projection?

In postfetal life the upper lateral cartilages are attached closely to the nasal bones in a unique way in that the cephalic margins of the former are intimately applied to the under surface of the latter. However, the remainder of the periphera of the upper laterals are more loosely connected by fibrous tissue to the pyriform margins of the maxillae, laterally, and to the cephalic margins of the lower laterals, caudally. This type of architecture of the upper lateral cartilages permits them to be pushed by growing adnexa in a ventral direction readily, less so caudally, and not at all cephalically. In other words, it allows the underlying quadrilateral septal cartilage to grow and expand ventrally and caudally without offering any resistance.

As we pointed out in another communication,[5] the lobule of the nose (including the lower lateral cartilages and membranous septum) with its fan-like attachment to the bony pyriform margin of the maxilla can be compared architecturally and in its mode of action to that of a moat bridge; the pivoting points for motion of the lobule are at its flanks, so that it can be easily raised and/or lowered. This arrangement, therefore, favors the growth of the underlying quadrilateral septal cartilage ventrocaudally without offering any resistance to its growth. In fact, bulbous tips or nonprojecting tips are frequently the result of either hypertrophied or underdeveloped quadrilateral septal cartilages, as the case may be.

B. Clinical Evaluation of the Embryology: The lateral cartilaginous walls help to maintain the continuous growth of the cartilaginous portion of the nasal profile as well as to allow for required movements of the nose during nasal respiration. Further, the moat bridge attachment of the lower lateral cartilages provides for recoil action in the event of trauma to the nasal tip, starting with the very delivery through the birth canal and lasting through all of life. Finally,

these cartilages favor the ventrocaudal growth of the septum.

The Septal Partition

The septal partition includes the septal quadrilateral cartilage, vomer, maxillary spine, and crest.

I. Septal Cartilage and Perpendicular Plate of Ethmoid.—A. Embryology: The nasal septum starts out as a thick membranous structure. Soon two plates of mesenchymal condensation (the precartilaginous stage) become evident. By the middle of the third month of fetal life, cartilage extends into the septum from the body of the sphenoid. Two cartilaginous plates are thus formed, each in juxtaposition to the other, which usually fuse into one. At this stage, the septum is beginning to thin out.

Further thinning takes place during the first year of postfetal life due to ossification beginning in the cephalic portion of the cartilaginous nasal septum which forms the lamina perpendicularis of the ethmoid, and this ossification extends into the crista galli and the cribiform plate.

The perpendicular plate does not fuse with its vomerine support until 40-45 years of life.

The nasal cartilaginous septum (quadrilateral cartilage) is surrounded by the end of the first year of postfetal life by the following bones: the frontal and the ethmoid above; the sphenoid behind; the vomer and the maxillary crest and spine below. These bones prevent the growth of the septal cartilage in their direction. However, at this time in the ventrocaudal (anteroinferior) areas attachments to the septum are only cartilaginous in character. These cartilaginous attachments to the septum therefore permit the growth of the septum in a ventrocaudal direction.

B. Clinical Evaluation of Embryology:

1. A thickened septum could be of embryologic origin and not necessarily due to trauma. It may also be due to the presence of two cartilaginous plates which never fuse as occurs in the bifid nose.

2. The septal cartilage is probably the most important organ in the maintenance and growth of the profile projection of the cartilaginous vault in youth.

3. Therefore, removal of the septal cartilage in youth carries the danger of early cartilaginous vault saddling, plus the loss of the normal growth process of the nasal profile.

4. In the discussion of the vomer and maxillary crest and spine, further clinical references to the septal cartilage will be made.

II. Vomer.—A. Embryology: The vomer develops bilaterally on each side of the posterior inferior margin of the cartilaginous septum. It commences to ossify at the eighth week of fetal life as nuclei appearing on each side of the midline below the nasal septum. During the third month, the ossification of the vomer proceeds in a forward direction and invades the posterior end of the septal cartilage. Thus a deep groove forms in the vomer in which the septal cartilage is lodged. The bilateral plates of the vomer unite from behind forward at the expense of the wedged-in septal cartilage; fusion in the vomer is completed by the 13th to the 15th year.

The vomer in its growth helps project the overlying quadrilateral cartilage in a ventrocaudal direction.

B. Clinical Evaluation of the Embryology: Clinically, how does the vomer affect the growth and support of the overlying septum?

We all have observed adults whose septal cartilage is found lying to one side of the vomerine groove or even touching the nasal floor. In many such instances the traumatic deviation of the septum occurred in youth. Nevertheless, the ventral growth of the septum on its own inertia, in spite of its displacement from the vomerine groove, provided a continuous nasal profile growth. It is true that in some of these cases the profile projection was less than normal, and

some even had cartilaginous saddles. However, the growth of the septum in a caudal direction proceeded normally despite its being out of the vomerine groove.

The caudodorsal (anterior) end of the septum is only loosely placed in the vomerine groove for many years, while quite early, even in embryologic life, the posterior portion of the septum is rigidly fixed. This explains why when trauma is inflicted to the nose early in life the septal cartilage is so frequently dislocated anteriorly out of the vomerine groove and so rarely posteriorly.

Incidentally, in passing, we should like to point out that any attempt to straighten out the vomer in cases with premaxillary projections or raising a vomerine mucosal-periosteal flap in repair of cleft palates is dangerous for two reasons: first, the single center of ossification of the vomer can easily be injured, and, second, complete evulsion of the nonfused vomer might occur.

III. Maxillary (Nasal) Crest and Anterior Nasal Spine.—A. Embryology: The maxillary crest arises from the superior and mesial border of the processus palatinus of the maxilla at about the sixth week of fetal life. It starts to fuse at the eighth week from before backward with its fellow of the opposite side to form a ridge. Fusion is complete at about the 10th week of fetal life. A groove on its superior margin receives the inferior border of the vomer.

In front of its articulation with the vomer, the ridge rises higher to articulate with the septal cartilage. By so doing, the septal partition of the nose is completed.

The anterior end of the maxillary crest projects beyond the free border of the nasal notch to form the anterior nasal spine. The latter might aid or interfere with the projection of the septal cartilage at this point.

B. Clinical Evaluation of the Embryology:

1. Overdeveloped or underdeveloped maxillary spines can distort the nasolabial angle, making it either obtuse or acute.

2. Incomplete fusion of the maxillary crests lead to various palatine clefts.

3. In bilateral cleft palates, with failure of union between the maxillary crests and the vomer, some flattening of the nose occurs.

Summary and Conclusion of Embryology and Growth of Nose

The growth and projection of the bony vault depend on the nasal processes of the frontal, the nasal bones, and the nasal process of the maxillae. Interference with the ossification centers of the nasal bones or maxilla, due to poor embryologic development, trauma, or surgical procedures in youth, will result in deformities and poor projection of the nasal bony vault.

Further, from our review of the embryology and growth of the nose, it is our belief that the quadrilateral cartilage of the septum is the main anatomical organ that controls the growth of the ventral (profile) projection of the cartilaginous vault of the nose and, to a less extent, its caudal projection. The vomerine, nasal bones, perpendicular plate of the ethmoid and the maxillae play accessary roles.

Because the quadrilateral septal cartilage is the keystone in the development of the profile projection of the cartilagenous vault, dare we either chip away at it or remove large segments of it or even undermine it without fear of subsequent interference with the continuous profile growth of the cartilagenous vault in children?

Nasal Physiology

Besides anatomical considerations, there are physiologic considerations in septal surgery in children. More specifically, we are referring to the role of the air currents of the nose.

In 1948 [6] and, again, in 1957,[7] we presented our experiments in tracing these air currents.

By using filter paper, moistened with pH indicators and placing the moistened paper against the septum of the nose, color changes take place in definite areas of the filter paper on inspiration and expiration. The

course of the color change on inspiration indicated that the air currents started at the nares and then took a parabolic curve toward the roof of the nose and then slanted down backward into the choana. On inspiration, the course of the air currents was the same as on inspiration, only in an opposite direction.

The color changes that took place on the moistened filter paper were such that they were photographed in their natural colors and then were used in slides for projection purposes. It is to be noted that no particulate matter was used in these experiments. The air passing in and out of the nose containing the CO_2 and bicarbonates arising from expiration gave rise to the pH changes and consequent color changes on the filter paper.

[Particulate matter is present in smoke and powders which were previously used by most experimenters on cadavers, models, or humans in an attempt to trace the trajectory of air currents through the nose. Smoke and powders used in that manner did not reflect the course of the air currents. The fallacy of such experiments lies in the fact that particulate matter less than 0.5 mμ or greater than 5 mμ passing through a tubular system set up their own electropotentials and, therefore, deposit themselves on the walls of such tubes irrespective of the air streams present. Smoke and powders invariably contain all sizes of particulate matter, including some below 0.5 mμ and others greater than 5 mμ.]

In considering the trajectory of the normal air currents in the nose, it becomes apparent that deviations of the septum and external nasal pyramid in the caudal (inferior) and ventral (anterior or profile) areas of the nose can and do cause serious disturbances in nasal physiology. Contrariwise, deviations along the floor (dorsally) are less compromising in character. Therefore, before any decision is made with respect to surgery on the nasal septum, the character of the deformity and the site of the deviation must be considered *pari passu* with the foregoing discussion on the normal trajectory of the air currents of the nose.

If the deviation is principally along the vomerine ridge, surgery should not be performed on the child for two reasons: The first is that it does not play a major role in obstructing the flow of air through the nose. Secondly, one should try not to interfere with the growth centers of the vomer, if at all possible.

If the deviation and obstruction are along the caudoventral area of the septal cartilage, serious impedimenta are offered to the air currents on both inspiration and expiration. Here surgery will have to be instituted.

Type of Surgery

It must be stressed that the minimal amount of septal resection with or without replacement of cartilage should be performed in children. It is better to get only a 50% restoration of nasal function or, at the most, a 60% improvement than to do extensive undermining and resection of the septal cartilage with 100% restoration of function. If the latter is carried out with a large septal resection, it may lead to a subsequent poor projection of the cartilaginous vault in a ventrocaudal direction (Case 2) for the reasons previously cited in the discussion on the embryology and growth of the nose. Further, much resection of a septal quadrilateral cartilage in a child weakens a structural member which helps to dissipate forces delivered to the overlying upper lateral cartilages and thereby aids in the prevention of cartilaginous saddles.

If septal surgery is indicated, it is best to make several small vertical incisions (Case 1), each just caudal to any buckled area of cartilage, than to make one long incision (ventrodorsally), as is customarily done in submucous resection with consequent extensive undermining of the mucoperichondrium. This is advocated so that only a minimum of cartilage will be removed in the first place and, secondly, that a minimum of interference with the nourishment

of the remaining septal cartilage will take place.

We feel it is better to do a partial and incomplete submucous resection in a child and defer further submucous resection, if it is necessary, to a time when the child's nose has attained its maximum growth—age 14 or more.

Where the cartilaginous septum is dislocated from the vomerine groove and it is causing obstruction to breathing, if it can be replaced into the groove without too much manipulation, it is the method of choice. This would reconstitute the normal anatomy and somewhat normal growth processes could be reasonably expected from this procedure.

The possibility that resected septal cartilage in children is replaced by the growth of new cartilage is open to question. The only place from which the new cartilage could arise would be the mucoperichondrium in the septal-free area or from the margins of the remaining septum.[8] We have had an experience (Case 2) which might indicate such a possibility. This question cannot be categorically determined by one instance. Further experience might indicate it to be so. Presently we feel that rebirth of cartilage rarely ever takes place, if at all.

Where there is a combination of a bony vault deformity in association with a septal obstructing nasal deformity in children, we prefer, first, to reduce the fracture of the bony vault by the closed method. If, without too much manipulation, the dislocated septum can be replaced into its proper position, it is done at this time. If the septal correction is not successful at this time, a minimum of three weeks is allowed to elapse before performing septal surgery. The type of septal correction has been described above and is performed under general anesthesia in addition to local with procaine and epinephrine. If infection (septal abscess) intervenes, a three weeks' waiting period after the infection has subsided is also required before septal surgery is instituted.

Further, we feel that a rhinoplasty with open reduction is rarely, if ever, indicated in children for fractures involving the nasal bony vault because most can be reduced by the closed method manually. Moreover, the single centers of ossification of each nasal bone may be more easily injured by open reduction than by closed reduction. Regardless of the exact location of the ossification center in the nasal bone, injury to it could hardly be avoided during rhinoplasty or open reduction considering the amount of elevation of skin and periosteum that is needed for proper manipulation compared to the small size of the nasal bones in children.

As for any external deviation of the cartilaginous vault not contributing to poor nasal physiology, we defer any corrective measures to the minimum age of 14 years to allow for further projection of the profile via the normal growth processes. It is felt that separating the upper lateral cartilages from the septum or tearing the fibrous attachments of the upper lateral cartilages at the pyriform margins of the maxillae may subsequently interfere with the forward projection of the upper lateral cartilages.

Even though we may defer operation on the external deviated cartilaginous vault not causing nasal obstructive breathing, we do not hesitate to operate upon the obstructing septum, if it is necessary, for restoration of normal nasal physiology.

Report of Cases

CASE 1.—A white boy, aged 9 years 9 months, was struck on the nose by a baseball on April 27, 1957. Bleeding occurred from the nostrils as well as from a small laceration over the bridge of his nose slightly to the right side, and also from a superficial laceration over the dome of the right lower lateral cartilage. Physical examination disclosed a markedly swollen nose with small superficial lacerations in the areas indicated in the history. Both nasal bones were fractured and crepitus was elicited. The nasal bones were shifted to the left. Both nasal chambers were filled with blood clots. There was a laceration of the septum on the right side. With the use of intra-

nasal local anesthesia, the fractured nasal bones were easily reduced to their normal position by the closed manipulation method. Subsequent examinations revealed a markedly deviated septum to the right in the ventral area and in the ventral and slightly below the ventral areas on the left side. On May 29, 1957, all the external edema had subsided. The nasal bones were in a normal position. The cartilaginous vault was widened and its profile depressed approximately 2 mm. below the level of the bony nasal profile. The latter resulted from a marked deviation of the septum which was principally located along the ventral margin of the septum. It was deviated to the right and left, the deviation on the left being somewhat more dorsally placed than on the right. The child had complete nasal obstruction on inspiration and expiration on both sides. This was accompanied by a story of severe snoring. On this date, he was admitted to the North Shore Hospital, Long Island, where, with general anesthesia and with some local infiltration of procaine and epinephrine, a unilateral incision was made on the right side of the septum for about 5 mm. in a ventrodorsal direction and placed just caudally to the angulated deviation on that side. A button of cartilage consisting only of the knuckled area was then removed by the submucous route. Similarly, two other ventrodorsal incisions were made on the left side of the septum, each just caudally to buckled areas (not opposite the vertical incision on the other side), and then two small buttons of cartilage were removed. Even though at the end of the procedure there was an obvious 2 mm. depression and a widening of the cartilaginous profile, no cosmetic surgery was instituted at this time. He was discharged on June 1, 1957, having made an uneventful recovery.

Examination on Sept. 7, 1957, disclosed a well-healed septum. The patient is breathing well, and the snoring has abated. There still remains a slight diminution of the nasal airway on the right side and slightly more on the left side of the nose. The improvement obtained is estimated to be about a 60% restoration of function, enough to satisfy both child and parents.

Comment.—1. Several vertical incisions, small in size, were made into the septal mucous membrane followed by limited elevation of the perichondrium in each instance. This is our modified type of submucous resection.

2. The cartilaginous vault still remains widened and its profile projection depressed approximately 2 mm. below the level of the bony nasal profile. The parents have been told that any future surgery on the septum as well as the cosmetic surgery will be deferred until such time as the nose has attained its full growth.

CASE 2.—A white boy, aged 9 years, was admitted to the Mercy Hospital, Springfield, Mass., on Nov. 2, 1953, with the following history: The chief complaint was nasal obstructive breathing which followed an injury to the nose about a year prior to his admission. Physical findings: The septum was deviated to the right and left with a subluxation of the caudal end causing the obstruction. On Nov. 24, 1953, under endotracheal anesthesia, most of the deviated quadrilateral cartilage was removed by the typical submucous route. The piece of cartilage removed was tongue-shaped in character, leaving substantially a frame of cartilage in the dorsal, caudal, ventral, and cephalic areas. Follow-up examination four years later, on Sept. 21, 1957, at the age of 13, revealed the following: Externally, the nose is straight. The bony vault is well-projected for his age. However, the cartilaginous vault, starting at the caudal end of the nasal bones, has failed to reach the same level of projection as the nasal bones. The lack of projection in the cartilaginous vault area takes the form of a slight curve, from the profile view, with the greatest fall-off at the distal end of the cartilaginous vault. Extranasal examination, however, does not reveal a saddle. Intranasal examination disclosed the left wing of the vomer slightly deviated on the left side, not enough to interfere with ventilation or drainage. The septum is straight. The area from which the septal cartilage was removed is so resistant to the nasal probe that cartilage appears to have regrown in this area. Nasal function in general is good.

Comment.—1. Externally the profile cartilaginous dorsum has not projected itself as much as the bony dorsum. It can be assumed that the bony dorsum, which was not operated upon, continued to project itself at its normal rate. However, the large amount of septal cartilage which had to be removed at the age of 9 years for restoration of nasal function interfered with the normal growth of the cartilaginous profile.

2. Regrowth of cartilage in the area of the septum from which it was previously removed is open to question for the following reasons: It should be remembered that the septum normally arises as two thin plates in juxtaposition to each other, which

later fuse or never fuse as in the case of the bifid nose. It is therefor possible that there remained after septectomy in this young child a very thin plate of quadrilateral cartilage which was not identified as such at operation. The difficulty in recognizing these thin cartilaginous plates is multiplied because such infant septal cartilages have marked glistening surfaces which can be easily mistaken for mucoperichondrium. Even during routine submucous resections in the adult we have frequently encountered the septum to be comprised of two plates of cartilage. In some of these instances one plate was straight and paper-thin and was therefore left in situ; the other, being thickened and deviated, was removed.

Case 3.—A white girl, aged 5 years, was admitted to the Mercy Hospital, Springfield, Mass., on June 8, 1951, with a chief complaint of difficulty in breathing through the nose and an external nasal deformity. At the age of one and one-half years, she fell and fractured her nose. Physical examination revealed a marked deviation of the nasal septum with subluxation of the caudal end almost completely obstructing both nasal chambers. Externally, there was a cartilaginous saddle and a drooped tip.

On June 8, 1951, with the patient under general anesthesia, a Freer type of submucous resection was performed. Three small pieces of deviated cartilage were removed. After this procedure, the nasal breathing was much improved.

Two months later, she was in an auto accident and once again injured her nose. Thereafter, the septum was displaced again, causing complete obstruction to the right side of the nose. Externally, the nasal bones appeared to be separated. In October, 1951, examination revealed a bony hump and a cartilaginous saddle.

On Nov. 7, 1954, the findings were the same as in October, 1951. However, because the nasal breathing was so poor at this time, she was readmitted to the hospital and operated upon with the use of intratracheal anesthesia. A submucous resection was performed. The deviated septum was removed, and several straight small pieces of pickled cartilage were inserted between the septal flaps. The saddle was not corrected at this time. Examination on Sept. 21, 1957, disclosed the nose to be in the midline with a small bony hump and a cartilaginous saddle. The cartilaginous vault was widened. Intranasally, the septum was straight, and the nasal breathing was good.

Comment.—1. Several small pieces of cartilage were removed at the first operation, with a good functional result, the typical ventral-dorsal incision in the septal mucous membrane being used. At the present time, for the same type of pathology, we no longer use the single ventral-dorsal incision, but, instead, small vertical incisions, each just caudal to and in juxtaposition to the areas of buckled septum, to allow for a minimum of mucoperichondrial elevation. By so doing, we feel that the least amount of interference with the nourishment of the remaining septal cartilage takes place. Secondary trauma to a previously operated upon septum with wide undermining of the mucoperichondrium in a child can contribute to weakness of the septum and its inability to withstand external trauma.

2. At the second operation, because more septal cartilage had to be removed in order to reestablish nasal breathing, pieces of pickled cartilage were inserted to help maintain the septal strength. Although this procedure has some value in this respect, it contributes little to the growth of the septum or to the profile projection.

Summary

The problems of the deviated obstructing nasal septum (recent or old), with or without external nasal deformity, in the child is approached by first discussing the development of the nose. Special emphasis is placed on the relative importance of each of the component parts of the nose in the maintenance of the continuous growth of the nasal profile. Pertinent physiology of the nasal air currents is discussed.

Indications for surgery of the obstructing nasal septum are considered. A new type of septal surgery is advocated for deviations of the septum in children. A discussion of the plastic surgical problems and their management in cases which have in addition an external cartilaginous vault bony deformity, is included. Illustrative cases are included.

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Peristapedial Pathology and Otitic Reinfection

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The human hearing organ can undergo microscopical examination in its entirety only once, and this post mortem. To observe intermediate steps of a pathological process one has to turn to animal experimentation. In aural diseases which lead unavoidably to the end, the condition in which the ears are found corresponds to an irreversible stage. An intermediary state of ear pathology is seen only when the specimen originates from a patient who died because of an extra-aural illness; otherwise intermediary stages have to be reconstructed largely by conjecture or by forming an arbitrary sequence of findings as obtained from different individuals.

Residua after a bout of otitic infection are considered as stagnant, unchanging. Yet this state is challenged by every new infection.

To gain an approach to the problem on an experimental basis one has to know that a large percentage of the current laboratory animals show spontaneous, i. e., nonexperimental otitic lesions, mostly originating from nasal and paranasal infection, which leave conditions of a fairly well-known nature. The present study proposed to take the characteristic organized residua in the middle ear as zero line and to create, by artificial impact, lesions of undoubtedly recent nature. An interpretation then becomes possible which opposes the result of the known, recently administered experimental attack against the previously existing situation.

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The material forming the basis of these investigations was compiled from a wide variety of sources, while the demonstrated pictures, for the sake of uniformity, originate from experiments with poisoning by fluoroacetate and by arsenic.

The histological details combine to create the picture, as to the residua, of a chronic suppurative productive (metaplastic) otitis media complicated by a collateral serous or serofibrinous otitis interna. Against this background are projected the products of the new, experimentally inflicted damage.

Hemorrhage and abscess formation dominate the newly created situation. The old organized residua are either able to withstand the new impingement, in which case the two pictures are juxtaposed, or the acute abscess breaks into the old product and a fresh sequence of pathological events starts. With colliquation of organized bands and fibrous masses, motility of the ossicular chain, severely handicapped by the previous organization, may be at least to a certain degree restored.

It has been shown that the important process of *reinfection* can be reproduced in animal experimentation. The method applied here was chemical impact. The recent attack can destroy the entire soft tissue apparatus of the middle ear, including fibrous bands and fibrous sheets.

The old accumulations show disastrous effects before all when they reach the oval and the round window. The stapes is embedded solidly among fibrous cushions, while around the ossicular articulations fibrous sheets like plaster casts are formed, producing immobilization up to the highest degree.

Destruction of the synechiae which are an otitic aftermath by injecting colliquating fluid into the middle ear was given up after several trials in the past as lacking any ap-

preciable success. Surgery—tympanoplastic measures—is used to correct the conditions caused by the products of inflammation. Yet nothing can change the picture so radically as a reinfection, after which new organization may appear, possibly in a slighter degree. Clinically a corollary is offered by the patient with otitic residua who reports that whenever his ear starts to discharge again his hearing improves.

Different factors—bacterial, viral, or chemical—leave different situations behind which vary, e. g., according to the various species of bacteria. A new invasion in case of reinfection does not necessarily result in lesions similar to the previous one. Although the new invasion might result in damage after healing, this might be of a different and possibly less severe character than the one left in the wake of the initial attack.

Summary

The pathological picture as caused by chronic organizing processes can be sufficiently demarcated to project against this background lesions left by a recent, experimental attack. Discrimination between the two sets of products may serve as a starting point for further analysis.

Clinically, converting a rigid static concept into a dynamic concept, one will have to remember the perpetually changing nature of residual products instead of considering them to be definitive and immutable.

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Recruitment Measured by Automatic Audiometry

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Introduction

Because loudness recruitment has assumed significant clinical and diagnostic importance, many tests have been devised to demonstrate its presence. One such test involves automatic audiometry. In 1947, when Békésy [1] first described his automatic audiometer, he also suggested that the amplitude of excursions of the audiometric tracing gives a measure of the difference limen (DL) for intensity. It was further reasoned that if recruitment were the result of an abnormally rapid growth of loudness, this rapid growth should be the result of diminished DL's. Therefore, diminished excursions in automatic audiometry have been taken as direct indications of the presence of recruitment.

The identity of diminished excursions on the automatic audiogram with DL's and recruitment has been supported by such authors as Reger,[9] Lundborg,[4] and Meurman.[5] On the other hand, Palva,[6] one of the more prolific critics of the measurement of recruitment by automatic audiometry, has suggested that the size of the excursion is not a measure of the DL, but rather of the rate of growth of loudness, and psychologists have recognized for some time that there is no direct relationship between size of a DL and the measure of loudness experience.

In 1954, Hirsh, Palva, and Goodman [3] argued that, on the basis of the loudness-function data of Stevens and Davis,[11] loudness in the normal ear increases more rapidly at low frequencies than at high frequencies; however, Riesz [10] showed that DL's at low frequencies are greater than those at high frequencies. Therefore, Hirsh, Palva, and Goodman concluded, the DL cannot be a measure of recruitment.

The above studies and speculations have been concerned with the relation to recruitment of width of threshold variations in automatic audiometry as a function of appreciation of loudness growth with change in intensity. The standard, or decibel, attenuator used in connection with the automatic audiometer has the characteristic of providing equal decibel changes of physical intensity of a stimulus with equal angular rotation of the controlling mechanism. The experience of loudness change, however, is different for equal intensity changes depending upon the level above threshold of the sound. This is equally true for the size of the DL. In view of this, it could be that, since the normal ear shows smaller DL's at intensity levels above threshold than at threshold, the narrow excursions shown by a recruiting ear, if such are correlated with size of DL, merely reflect the fact that the ear is operating like a normal one at a level above normal threshold. It would seem desirable, therefore, to investigate the difference between normal and recruiting ears with respect to perception of loudness growth as a function of loudness change rather than intensity change as measured by variation around threshold with the automatic audiometer.

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In 1955, for the purpose of making further measurements of the loudness function, Poulton and Stevens [8] designed what they called a "sone potentiometer." The departure of their apparatus from the standard attenuator lay in its attenuation characteristics. With the sone potentiometer, or loudness attenuator, in the circuit, equal increments of rotation provide approximately equal increments of perceived loudness, based on the loudness function curve for 1000 cps. Essentially, then, whereas the standard attenuator produces logarithmic intensity growth (which is linear in terms of decibels) with rotation, the loudness attenuator in terms of present application, produces a varying intensity growth with rotation. The loudness attenuator of Poulton and Stevens and a 1956 revision by Stevens and Poulton [12] are the only two such instruments reported in the literature to date.

In view of the preceding discussion, the basic questions to be answered by the present investigation are the following:

1. Do recruiting ears show narrower threshold tracings than do normal ears when subjected to automatic audiometry under standard conditions, that is, with the stimulus varied so that equal increments of intensity are produced by equal increments of attenuator rotation?

2. When voltage is varied to produce unequal increments of intensity with equal increments of attenuator rotation, do recruiting ears respond differently than they do under standard conditions of automatic audiometry?

3. Do people with recruitment respond differently than those with normal hearing under the above conditions?

4. Does the individual observer respond in automatic audiometry in accordance with his DL's for the frequency, or in accordance with his loudness experience?

Experimental Design

Subjects

Normal Hearing Population.—Ten subjects (four men, six women) with an age

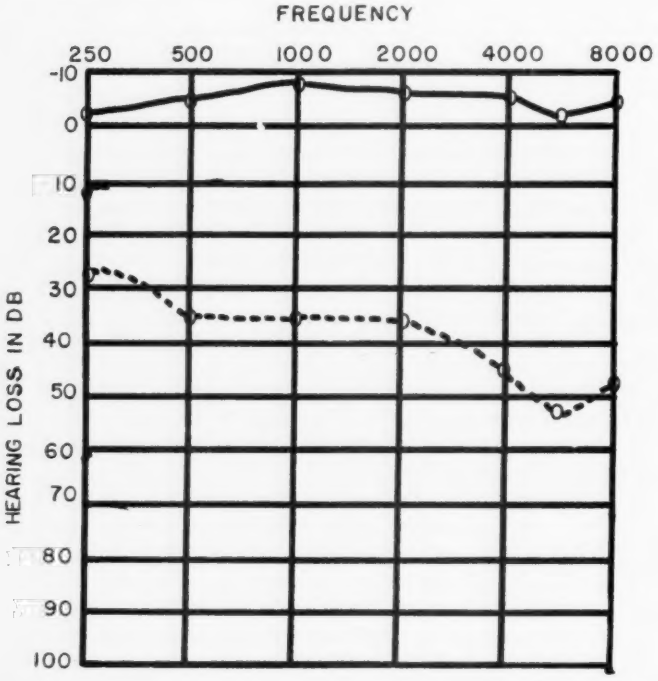

Fig. 1.—Composite audiograms of normal and experimental groups. The normal group is represented by the solid line and the experimental group is shown by the broken line.

range of 26 to 38 years and a mean of 30.0 years were chosen to comprise the normal group. Criteria for inclusion were as follows: no previous experience with automatic audiometry in any form, and at least one ear with a threshold no higher than 15 db. (re normal) at the frequencies to be tested.

Population with Recruiting Ears.—Twelve ears were chosen for inclusion in the recruiting group, representing an age range of 25 to 71 years with a mean of 44.9 years. Each subject had an otological diagnosis of Ménière's disease in at least one ear, and demonstrated recruitment at at least one frequency by the appropriate standard loudness-balance test. No subject had had previous experience with automatic audiometry. Figure 1 shows the composite audiograms of the normal and recruiting ears.

Apparatus

For pure-tone audiometric testing, one of two standard audiometers was used: an Audivox 7-BP and a Maico H-1B. The latter instrument was used for all loudness-balance procedures. All preliminary threshold and recruitment tests were performed in sound-treated chambers.

The automatic audiometer was constructed under the direction of Dr. Merle Lawrence in the Physiological Acoustics Laboratory of the Department of Otolaryngology at the University of Michigan. The loudness attenuator was custom-built in the Laboratory according to the circuit given by Poulton and Stevens.[8] The automatic audiometer is shown by block diagram in Figure 2. The oscillator used as a source of the pure-tone stimuli was a General Radio, Type 1304-B. The attenuator in the circuit was a Daven, Type T-690-C. The earphone was a Permoflux PDR-8. Graphic recordings were made by a Sandborn Electric Stylus on Sandborn heat-sensitive Permapaper.

The input voltage to each variable attenuator was adjusted to compensate for insertion loss and to place normal thresh-

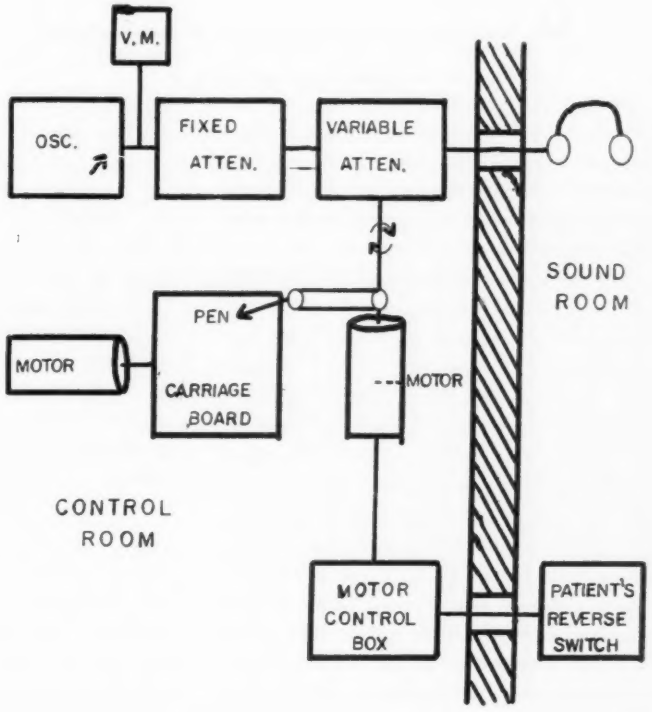

Fig. 2.—Block diagram of the automatic audiometer.

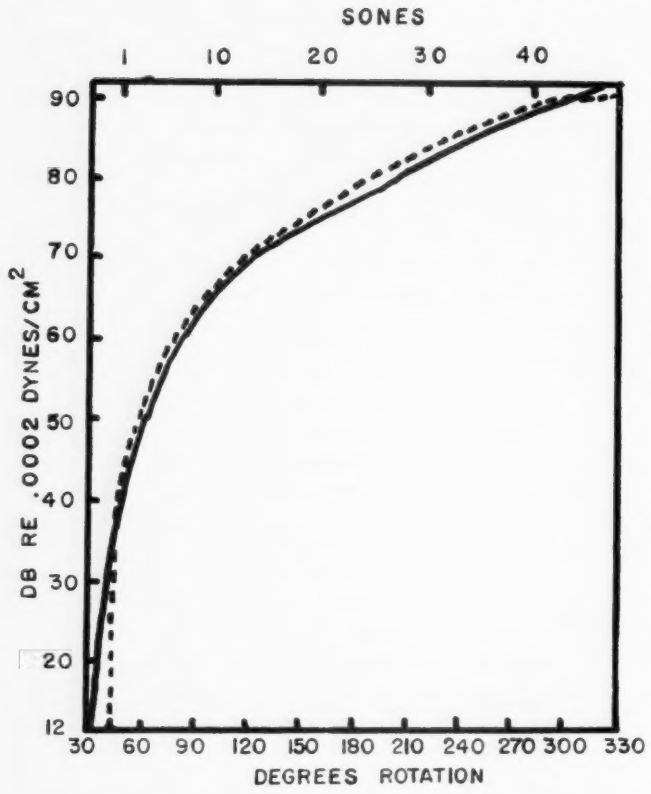

Fig. 3.—Output characteristics of the loudness attenuator compared with the loudness function for 1000 cps. The solid curve represents the loudness attenuator and the broken line represents the loudness function. The upper abcissa refers to the loudness function; the lower, to the attenuator curve. Relative placement of the two scales is chosen arbitrarily for ease of visual comparison of the curves.

old at the low intensity end so that maximum range could be obtained. Appropriate voltages were found to be 10 volts for the standard attenuator and 1 volt for the loudness attenuator.

Figure 3 shows the output characteristics of the loudness attenuator compared with the loudness function curve for 1000 cps, according to Stevens and Davis.[11] Evaluation of this figure shows that the loudness attenuator of the current study approximates the loudness function to a reasonable degree.

The carriage board was operated at a speed of ½ in. per minute. The heat-sensitive paper was prepared by marking a series of seven columns, each of which was 1 in. wide. Thus, a two-minute sample of each frequency was obtained with the column borders serving as signals to the experimenter to adjust the frequency of the stimulus after the required time interval per frequency had elapsed.

The standard attenuator provided essentially continuous attenuation at a rate of 2.5 db. per second. The motor speed governing the loudness attenuator was the same as that which produced the above figure for the standard attenuator.

The patient's switch was so constructed that intensity was caused to decrease when the control button was manually depressed, and intensity increased when the button was released.

Procedure

Each subject was given a standard puretone audiometric test to determine hearing acuity at the frequencies to be investigated later: 250, 500, 1000, 2000, 4000, 6000, and 8000 cps. In addition, the hard of hearing subjects were given loudness-balance tests

to determine the presence of recruitment at as many different frequencies as could be tested by either the Fowler binaural method or the Reger monaural method.

Collection of Experimental Data.—Automatic audiometric tests were performed with the subject seated in a sound-insulated room in the Physiological Acoustics Laboratory. The automatic audiometer was located in an adjoining chamber which had a soundproof window into the testing area. The subject was given instructions, essentially, to cause the stimulus to cross his threshold repeatedly until the conclusion of the test.

The oscillator was set at 250 cps, and the subject listened to this frequency for a period of two minutes, during which time he controlled the attenuator so that the tone would vacillate continuously across his threshold. At the end of two minutes, the oscillator was manually adjusted by the experimenter to the next higher frequency without interrupting the test, and the subject continued his task. The seven frequencies were thus tested. At the conclusion of the test, the subject was allowed to relax for approximately five minutes while the experimenter substituted the second attenuator. Half of the subjects, on an alternating basis, were tested with the loudness attenuator first, and half with the standard attenuator first. After the necessary adjustment the subject repeated the test with the other attenuator. Alternating the order of presentation of the two attenuators was intended to avoid the bias which might have resulted from a consistent order of presentation, coupled with possible learning effects. A discrete frequency presentation was chosen in preference to a continuous frequency presentation to facilitate the measuring process which was to follow.

Treatment of Experimental Data.—The attenuation rate of 2.5 db. per second of the standard attenuator corresponded to a linear measurement on the recording paper of 1 db. per millimeter. Width of excursions was determined by selecting the middle ½ in. (corresponding to one minute of stimulation) of each frequency sample. Linear measurements were made in millimeters for each descending record trace during the chosen interval (to a maximum of 10 measurements per frequency for ease in arithmetic manipulation), and the mean width per frequency was obtained. Because of the correspondence of 1 db. to 1 mm., widths could easily be recorded in terms of decibel excursions.

Although selection of excursions to be measured followed the same pattern for the loudness attenuator aspect of the data collection as for the standard attenuator aspect, there was no simple linear relationship between millimeters and decibel excursions. Therefore, as soon as the loudness attenuator test was completed, the carriage board, with recording paper intact, was run manually under the stylus at a predetermined degree rotation setting in order to produce a horizontal line of orientation on the recording sheet. With this orientation, using the scale of 2.3 degrees per millimeter, measurements were made of the extent of the excursions in terms of degrees rotation of the attenuator. The mean range of degrees traveled was converted to decibel measurements from the data shown in Figure 3.

In the above manner, the mean width in decibels of the threshold tracings for each frequency for each subject was obtained. These data were collected independently for measurements made with the standard attenuator and with the loudness attenuator in the circuit.

Results

Of a maximum of 308 possible observations (seven frequencies by 22 subjects under two conditions), 23 observations were not collected for reasons such as severity of hearing loss at certain frequencies, temporary lapses of attention by the subject, or temporary unforeseen interruptions of the test. In order to facilitate the analysis of variance, the number of observations in each group under each testing condition had to be made equal. This was accomplished by accepting the set of data with the minimum

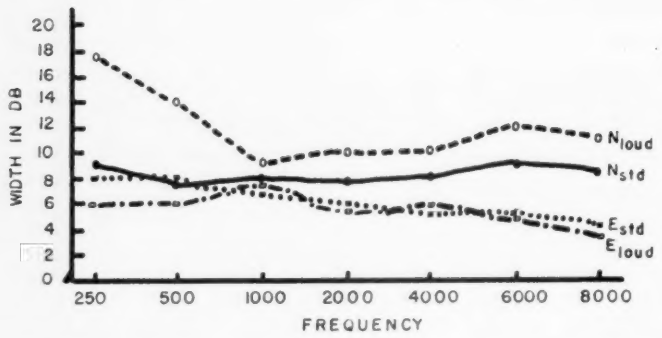

Fig. 4.—Mean width of threshold excursions in decibels produced by two subject groups under two conditions of automatic audiometry at seven different frequencies.

N_{std}—normal group, standard attenuator

N_{loud}—normal group, loudness attenuator

E_{std} — experimental group, standard attenuator

E_{loud} — experimental group, loudness attenuator

number of observations (normal subjects, standard attenuator) as the standard, and adjusting the other groups of data to conform to this standard by discarding certain data on the basis of a table of random numbers.

The remaining data, a total of 252 observations, are presented in graphic form in Figure 4, which figure shows the mean width of threshold excursions at each of the frequencies tested for both groups and both conditions. The difference between subject groups, conditions, and frequencies, plus the several interactions, were analyzed through an analysis of variance as described by Edwards.[2]

Excursions Measured in Decibels.—Consideration of the data reveals that under conditions imposed by the standard attenuator, the experimental group tended to produce diminished excursions in the higher frequencies, whereas the normal group showed the same or larger excursions in the same higher frequencies. This is consistent with results from earlier reports on recruiting ears, and is supported statistically. The response of the normal group to the standard attenuator shows acceptably close agreement with similar data published by Palva.[7] It having been established that the two groups differ in response to automatic audiometry under standard conditions, it is interesting next to inspect the differences in response to similar audiometry with the loudness attenuator in the circuit.

Comparison of the "N_{std}" curve of Figure 4 with the "N_{loud}" curve shows that the normal subjects, as a group, produced larger decibel excursions with the loudness attenuator than with the standard. This is supported statistically. The extreme differences in the low frequencies will be discussed later, but it can be seen that the two curves run approximately parallel from 1000 to 8000 cps, with the loudness attenuator excursions averaging 1.8 db. greater over the parallel range. This situation may be taken as a standard against which to compare results obtained from recruiting ears. Because the subject's appreciation of loudness must remain the same at threshold from one test to another, the above differences can be accounted for on the basis of a different rate of change of intensity from one attenuator to the other. Knowing how the normal group reacted to the loudness attenuator, we may now inspect the responses of the experimental group.

The experimental group is seen to have behaved differently from the normals on the basis of the statistically significant difference between subject groups. Introduction of the loudness attenuator into the testing situation made relatively little difference in the size of threshold excursions demonstrated by the recruiting ears compared with the increase in threshold excursions in decibels elicited from the normal group, as noted above. Over the middle and high frequency range, loudness attenuator excursions averaged 0.6 db. less than standard excursions over the same range.

The loudness attenuator, it will be recalled, varied voltage so that intensity grew

approximately in accordance with the normal loudness function. Comparison of the response of normal ears to the loudness attenuator (N_{loud}) with the response of the recruiting ears to the same attenuator (E_{luod}) reveals that threshold tracings shown by the recruiting ears were an average of 4.7 db. smaller than were threshold tracings shown by the normal ears over the middle and high frequency ranges. Clearly, then, application of the loudness attenuator did not affect the two groups of subjects in the same manner, indicating that the growth of loudness was different in the experimental than in the control group.

Factors of decision time and reaction time were assumed to be constant for both groups and had to be ruled out as significant in determining the above differences; however, a possible clue was revealed in considering the average thresholds of the groups, as shown in Figure 1, plus the output characteristics of the loudness attenuator, as shown in Figure 3. The average threshold of hearing of the normal group was 45.5 db. lower than that of the recruiting group. This meant that the normal group was functioning over a section of the output curve of the loudness attenuator where the slope was particularly steep, and thus intensity was growing very rapidly compared with the section over which the recruiting ears were operating. It was speculated that perhaps reaction time assumed a relatively high percentage of total excursion time for the normal ears in this condition. In other words, to the normal ears, intensity changed so rapidly that by the time the subject could make a decision about the appearance or disappearance of the tone and react accordingly, loudness would have changed far more than would have been the case had the subject operated over other sections of the output curve where intensity grew more slowly. This situation could have led to spuriously large decibel excursions.

To test the above possibility, 9 of the original 10 normal hearing subjects were submitted to a third condition of automatic audiometry approximately one week after the initial testing session. The conditions employed during this later aspect of the experiment were with the loudness attenuator in the circuit, but with a fixed attenuator, in the circuit immediately after the oscillator, set at 40 db. The rationale for this was that now the normal subjects would be forced to operate as if they had a 40 db. conductive hearing loss. This electronic "hearing loss" would place the responses of the normal group in approximately the loudness attenuator output range through which the recruiting ears had operated, thus alleviating the biasing effects of reaction time and decision time. Testing procedure and method of collecting data were essentially the same for this third condition as for the previous tests.

Figure 5 shows the results of the above method of subsequent testing. Average threshold tracings produced by the normal ears under conditions of the original procedure involving the loudness attenuator (conditions which we may now call

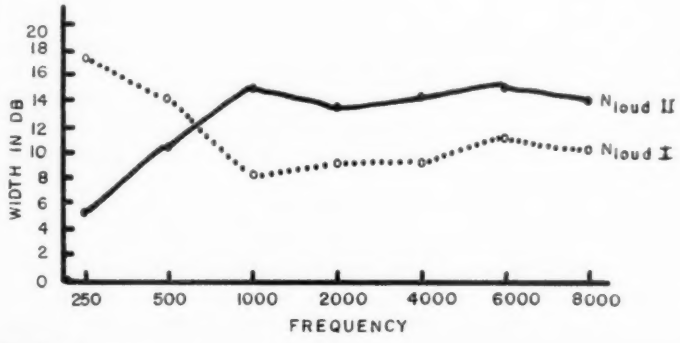

Fig. 5.—Mean width of threshold excursions in decibels produced by normal subjects under two conditions of automatic audiometry involving the loudness attenuator.

$N_{loud\ I}$—original conditions: corresponds to N_{loud} of Figure 4

$N_{loud\ II}$—loudness attenuator with 40 db. fixed attenuation prior to variable (loudness) attenuator

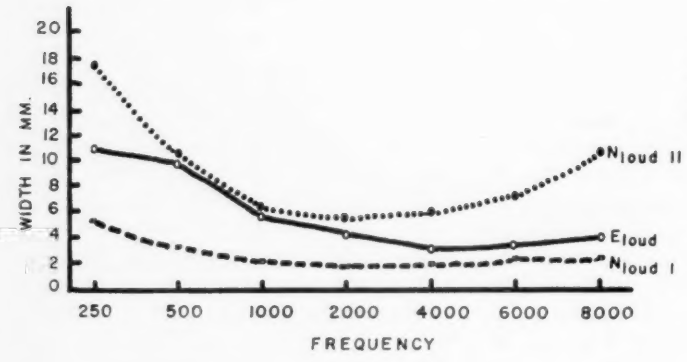

Fig. 6. — Widths of threshold excursions in millimeters for the two groups of subjects under conditions of the loudness attenuator in automatic audiometry.

$N_{loud\ I}$ — normal group, original measurement (see text)

$N_{loud\ II}$ — normal group, second measurement (see text)

E_{loud} — experimental group, loudness attenuator

"$N_{loud\ I}$" are shown in relation to results obtained from the subsequent testing procedure ($N_{loud\ II}$). The observations at the middle and high frequency ranges are shown to be indicative of larger threshold excursions under the revised testing conditions than was the case under the original conditions with the loudness attenuator by an average of 4.9 db. This may be interpreted as showing that when the potentially biasing factor of reaction time was made relatively constant between the two groups, the actual difference in responses of normal and recruiting ears was emphasized rather than reduced.

Excursions Measured in Millimeters.— Figure 6 shows the widths of threshold excursions in millimeters for the two groups of subjects under conditions of the loudness attenuator in automatic audiometry. It is seen that the normal ears produced very small millimeter excursions, but this was because of the very rapid growth of intensity produced by the loudness attenuator at the threshold of the normal group. When the normals were made to operate through approximately the same loudness attenuator output range through which the recruiting ears operated (where intensity grew more slowly), then the normal ears reacted more slowly than did the recruiting ears. In other words, the experimental group (E_{loud} of Fig. 6) reacted more rapidly to changes in "loudness" than did the normal group ($N_{loud\ II}$) under approximately the same conditions.

That the above reactions were to loudness growth rather than to DL's is seen by considering research data published previously. Riesz [10] showed that the ear responds to smaller DL's in the middle frequency range than at low or high frequencies. If automatic audiometry measured the DL, then the graphs of the excursions in decibels (Figs. 4 and 5) should show "saucer-shaped" curves indicating increased sensitivity in the middle frequencies. It is apparent that this is not the case.

Stevens and Davis,[11] on the other hand, have pointed out that the ear is more sensitive to loudness growth in the middle frequencies than in the high or low frequencies. If automatic audiometry measures sensitivity to loudness growth, then the graphs of excursions in millimeters (Fig. 6) should show the significant "saucer-shaped" curves. It is apparent that such is the case: the curves of Figure 6 show an increased sensitivity to loudness changes in the middle frequencies. The response to high frequencies by the experimental ears is a reflection of recruitment which shows more readily in the high frequencies (see next section). It is seen, then, that the automatic audiometer provides a measure of sensitivity to loudness growth, rather than a measure of the DL.

Comment and Conclusions

Clinical Implications

The preceding discussion of results was based entirely on group data with little attention to individual differences in response.

It behooves us, however, to examine more closely the differences which were noted among individuals of the same groups.

As an example, at 1000 cps under conditions imposed by the standard attenuator, the normal group displayed a range of threshold excursions of 5.6-12.0 db., while the recruiting ears at the same frequency displayed a range of 3.5-12.1 db. The degree of overlap cited is fairly typical of what is found at other frequencies when similar conditions are compared. This overlap means, then, that it is quite difficult to decide on the basis of a set of observations from a single subject to which group he belongs, unless the observations represent an extreme case in the group.

Palva [7] has advocated acceptance of a level of 5 db. as the critical criterion of recruitment in automatic audiometry; excursions narrower than this value are considered to be indicative of recruitment. If Palva's criterion is applied to the data of the present study, we may see that 95.2% of the observations by normals were wider than this critical figure; clinically, then, only 4.8% of the normals would show excursions so narrow as to be considered indicative of recruitment. This might be an acceptable margin of error for a clinical test of this type. On the other hand, only 32.1% of the observations of the recruiting ears were narrower than 5 db. This would mean that 67.9% of the tracings made by recruiting ears would be called nonrecruiting. This is obviously not an acceptable margin of error. Raising or lowering the criterion of 5 db. does not solve this problem.

Inspection of the mean widths of threshold tracings in decibels (Fig. 4) shows that under both conditions of automatic audiometry tracings produced by the recruiting ears get narrower as frequency is increased. This phenomenon does not occur with the normals and is taken as the "classical" sign of recruitment in automatic audiometry. When we inspect the extent of this narrowing for individual subjects, however, we find that when the standard attenuator is employed, the extent of narrowing from middle to high frequencies differs too slightly from the responses of the normals to be useful clinically. With the loudness attenuator in the circuit, a different situation is seen. Essentially, when, for an individual subject, the width of threshold excursion at 1000 cps is compared with that for 6000 or 8000 cps, a significant narrowing is seen. Therefore, it appears that the loudness attenuator provides a sensitive measure of recruitment in automatic audiometry when the narrowing of excursions from the middle to the high frequencies is taken as the criterion. For the loudness attenuator to be most useful in clinical procedures, it is desirable that the rate of movement of the recording stylus be divorced from the rate of change of the attenuator in such a way that equal decibel changes may be linear functions of distance in millimeters on the recording paper, as is the case when the standard attenuator is used. This flexibility would permit a direct measure of the width of threshold excursions without tedious measurement and computation.

For both clinical and research purposes it should prove interesting and valuable to construct a loudness attenuator which varies voltage so as to produce an output curve in the form of a "mirror image" of the loudness function. An attenuator of this type would produce equal increments of loudness with equal increments of rotation of the controlling mechanism so that the perception of loudness would be linear with rotation.

Theoretical Considerations

Loudness Attenuator Responses at Low Frequencies.—It was mentioned earlier that the normal subjects showed responses at low frequencies which were inconsistent with their responses at higher frequencies. To explain this apparent discrepancy, it is necessary to remember that the method used by Stevens and Davis [11] in defining the loudness function consisted in pooling data at 1000 cps furnished by a variety of experimenters. The loudness function at other

frequencies was defined by a series of experiments in which tones of various frequencies were equated with known loudnesses of a 1000 cps tone. Experimentally, it is possible that this relatively indirect method of determining loudness functions for frequencies other than 1000 cps may have introduced a second-order error, particularly at the low frequencies.

Moreover, the loudness function curves show that loudness grows more rapidly at low frequencies than it does at higher frequencies; therefore, it may be concluded that the loudness attenuator based on the loudness function for 1000 cps is inappropriate for the low frequencies.

Recruitment at Low Frequencies.—Most experimenters speak of recruitment as a phenomenon occurring exclusively at high frequencies. On the basis of Fowler's alternate binaural loudness-balance test, in the present study, recruitment was demonstrated at 250 cps by 5 subjects, and at 500 cps by 3 subjects, of the 12 subjects tested.

According to the concepts of the recruitment phenomenon which have been more or less widely assumed, it might be argued that the narrowing of threshold excursions (pointed out earlier) occurs only in the high frequencies because recruitment occurs only in the high frequencies. The question that we must ask, then, is whether recruitment is represented by an absolute narrowing of threshold tracings in automatic audiometry, or whether it is shown as frequency is increased. To answer this question, those data which were collected only at frequencies at which recruitment was actually demonstrated by the appropriate loudness-balance tests were selected from the complete set of data. The comparison thus obtained showed clearly that the narrowing effect is present even when recruitment has been shown as adequately for the low frequencies as for the high frequencies. Figure 7 shows the recording sheet obtained under standard conditions from a subject who demonstrated recruitment at all frequencies, thus illustrating the point just made concerning the lack of absolute narrowing but with an evident relative narrowing from middle to high frequencies.

Conclusions

The following conclusions appear justified within the limitations imposed by the procedure, apparatus, and subjects used in the investigation:

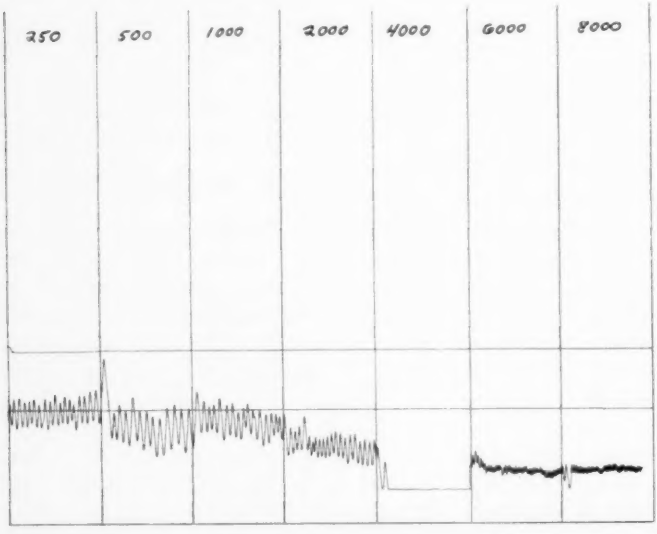

Fig. 7.—Recording sheet obtained from a subject who manifested recruitment at low frequencies as well as at high frequencies. Tracings were made under conditions of automatic audiometry with the standard attenuator in the circuit.

1. Taken as a group, recruiting ears give narrower threshold excursions than do normal ears in response to standard automatic audiometry.

2. Notwithstanding significant group differences, it is difficult to separate individual recruiting ears from nonrecruiting ears on the basis of standard automatic audiometry.

3. The use of the loudness attenuator in automatic audiometry provides a sensitive test of recruitment, using the criterion of a narrowing of threshold excursions from middle to high frequencies.

4. In automatic audiometry, subjects respond to growth of loudness, rather than to DL's.

Summary

Using a loudness attenuator which varies the voltage of a stimulus so as to produce unequal increments in intensity with equal increments of rotation, 10 normal ears and 12 recruiting ears were given automatic audiometric tests at seven discrete frequencies so that results obtained could be compared with similar data obtained through more standard means.

Analysis of the data by groups revealed that recruiting ears produced narrower threshold excursions at high frequencies than did normal ears under conditions of standard automatic audiometry. This effect was emphasized when the loudness attenuator was used. When the normal ears were retested in such a way as to induce them to operate through approximately the same range of the output curve of the loudness attenuator as the recruiting ears had used, the difference in width of excursions between the two groups was even more emphasized.

Notwithstanding the significant differences in group means, it proved particularly difficult to assign individual subjects to the normal or recruiting group on the basis of measurements from standard automatic audiometry. The loudness attenuator, on the other hand, provided data on the basis of which such assignment could be made with considerably more confidence. It was shown that defective ears manifest recruitment by a narrowing of threshold tracings from middle to high frequencies, rather than by an absolute diminishing, even in cases in which recruitment could be demonstrated at low frequencies by loudness-balance procedures.

Four general conclusions can be drawn from the study: 1. Taken as a group, recruiting ears give narrower threshold excursions than do normal ears in response to standard automatic audiometry. 2. Notwithstanding significant group differences, it is difficult to separate individual recruiting ears from nonrecruiting ears on the basis of standard automatic audiometry. 3. The use of the loudness attenuator in automatic audiometry provides a sensitive test of recruitment, using the criterion of a narrowing of threshold excursions from middle to high frequencies. 4. In automatic audiometry, subjects respond to growth of loudness, rather than to DL's.

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Subcutaneous Emphysema

A Complication of Tonsillectomy

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Subcutaneous emphysema is an uncommon but not a rare complication of tonsillectomy. Since 1910, when Parish [1] first described this condition, it has recurred periodically in the literature. I found a total of 22 reported cases. The following report is presented to bring attention to this alarming but rarely dangerous condition.

Report of a Case

An 8-year-old white girl was admitted Jan. 1, 1958, to Minneapolis General Hospital with a history of recurrent tonsillitis and otitis media for the past four years. Physical examination revealed small scarred tonsils and slight pharyngeal erythema. The left tympanic membrane showed scarring, and auditory acuity on the left was moderately diminished. Multiple shotty cervical nodes were present. The remainder of the examination was unremarkable.

On Jan. 2, 1958, under general anesthesia with use of thiopental induction, nitrous oxide and oxygen, tonsillectomy and adenoidectomy were performed. The tonsils were removed by dissection, and no significant injury to the tonsillar fossae was observed. Bleeding from the fossae was controlled by pressure and four plain absorbable surgical sutures.

Shortly after surgery the patient began to vomit and retch repeatedly, and her temperature rose to 102 F. She was placed on intravenous fluids and antibiotics. On the morning after surgery her left cheek was noted to be swollen, soft, and crepitant, and x-rays showed air to be present in the soft tissues (Fig. 1). After two days the vomiting and retching subsided, and the patient was able to retain oral food and fluids. The swelling remained unchanged, and no signs of infection appeared.

She was discharged seven days after surgery, with only slight decrease in size of the left cheek swelling (Fig. 2). By the 11th postoperative day the swelling had disappeared completely, and the patient had no further difficulties. At no time in the course of this illness had dyspnea, chest pain, or unusual chest findings been noted, and the swelling and crepitation remained localized to the left cheek only.

Fig. 1.—X-ray showing air in soft tissue of left cheek.

Comment

Review of the literature reveals that subcutaneous emphysema following tonsillectomy occurs under a wide variety of conditions. It appears in all age groups, both sexes, and under both local and general anesthesia. Onset is usually during or shortly after surgery but can develop, as it did in this case, as long as 24 hours later. Ordinarily no serious complications arise, but cases have been reported where respiratory embarrassment from mediastinal emphysema and subsequent pneumothorax presented a threat to life. No fatalities have been reported. Excessive trauma to the tonsillar bed during tonsillectomy has not been a significant factor in most of the reported cases. There is, however, a frequent incidence in reported cases of severe

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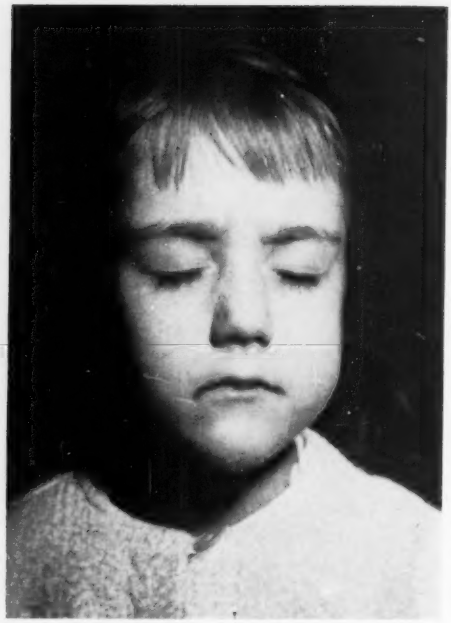

Fig. 2.—Photograph showing swelling of left cheek.

vomiting, coughing, and occasionally high gas pressures applied to the pharynx with anesthesia apparatus. The case reported in this article suffered protracted emesis beginning shortly after surgery and lasting about three days.

There are two mechanisms by which this complication may arise. The one that appears to account for most of the cases is brought about by pulmonary alveolar rupture, which in turn causes pulmonary interstitial emphysema that dissects its way to the mediastinum and neck. The emphysema may also extend retroperitoneally and to any subcutaneous area in the body. Proponents of this theory include Silverman,[2] MacCready,[3] and Keen.[4] The mechanisms of this process have been well worked out by Macklin and Macklin,[5] who have shown the emphysema in progressive stages by overinflating animal lungs. Pulmonary interstitial emphysema produced by increased pulmonary pressure associated with coughing, vomiting, and straining is clearly the cause of subcutaneous emphysema in the majority of cases. Silverman feels that every case of subcutaneous emphysema is secondary to alveolar rupture and that lateral chest x-rays are essential to demonstrate this process.

A second and less common mechanism is that of air dissection through a defective tonsillar bed. In the face of increased oropharyngeal pressure caused by vomiting and coughing, the air breaks through the superior constrictor pharyngis muscle and the buccinator fascia into the loose areolar tissue of the cheek and neck. Stein,[6] Knutson,[7] and Stevenson [8] have argued in favor of this mechanism. There have been a number of reports in the literature of subcutaneous emphysema of the head and neck that developed after trauma and surgery to the nasal or oropharyngeal mucosa. The emphysema usually follows increased nasopharyngeal pressure such as found in nose blowing. Edwards [9] cites an interesting case, reported by Turnbull in 1870, of a bugler who after having a dental extraction developed facial emphysema while blowing mess call. Stevenson mentions a notable finding in his case report, viz., that after the development of emphysema his patient had a frothy or bubbly saliva. The most conceivable source of this air would be the oropharynx or more specifically the tonsillar fossae.

I feel that the case of subcutaneous emphysema reported in this article was caused by air dissection through the tonsillar fossae, since the swelling was discrete and localized to the cheek. Also with the absence of symptoms and physical findings in the chest it is difficult to explain this on a basis of alveolar rupture and mediastinal emphysema.

The most logical answer to the question of etiology is that both mechanisms can cause subcutaneous emphysema following tonsillectomy. Well-documented cases that fit both theories are found in the literature, and adherence to one theory only seems untenable. It would appear, however, that the majority of cases are due to alveolar rupture

and mediastinal emphysema, and that the case presented represents an uncommon form of this complication.

Summary

A case of subcutaneous emphysema complicating tonsillectomy is reported. The incidence of this complication and predisposing factors are discussed. Two theories explaning this phenomemon are reviewed, viz., alveolar rupture with subsequent dissection into the soft tissues of the neck, and, secondly, dissection of air through the tonsillar fossae into the subcutaneous tissues. I feel that both mechanisms can, under separate circumstances, cause this complication.

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Nasal Sinus Diseases

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Nasal sinus disease occurs in children as frequently as it occurs in adults, but it is more frequently neglected in children. Any child who has a prolonged cold with a persistent bronchial cough usually reveals an infection of one or both maxillary sinuses, and with every established maxillary sinus infection there occurs accompanying infection of the ethmoid cells. Food allergies are particularly prone to underlie these infections, and the commonest offending foodstuffs are milk, eggs, wheat, chocolate, and corn. Often these children are pale and overweight, presenting pale swollen nasal membranes. The importance of diet and general treatment cannot be overemphasized. I do not hesitate to give these children at ages of 6 to 12 years ½ grain (30 mg.) of thyroid and 2½ grains (0.15 gm.) of Calcidin (a combination of iodine with lime and starch plus calcium iodate) (coated tablets), one of each to be taken before breakfast for a period of two weeks and continued if necessary. Occasionally, especially in the thinner and underweight child, the thyroid may produce disturbed sleep, necessitating its exclusion.

At the same time chloriodized oil (Iodochlorol) is injected by means of a double-curved cannula through the normal ostium. With care this can be done under local anesthesia in children 6 years or older. Only occasionally is a straight needle used under the inferior turbinate. If cooperation is impossible, a general anesthetic may be necessary for the injection, and if the case is old and of long standing, under the same anesthetic a large opening may be made under the inferior turbinate. At this time the tonsils and adenoids should be removed if indicated. In adenoidectomy, the palate should be retracted and the area carefully inspected for any remaining adenoid tissue. At the same time it is well to press the inferior turbinates laterally by means of a moderately curved closed hemostat.

The ethmoid labyrinth should not be disturbed at this time, for, as the antrum infection clears, the ethmoid infection subsides.[1]

Often it will be found that a child is consuming a quart or more of milk per day plus chocolate and soft drinks, all of which should be curbed. A good breakfast should be encouraged, since it is probably the most important meal of the day, following 10 hours without food. Fruit juice plus oatmeal with strained honey for sweetening offers an excellent start for the day. Few people are allergic to oats, and it is still the most potent food for horse and man. In the more acute cases the iodine is given two or three times a day, while not more than ½ grain of thyroid is used.

It is interesting that children from the age of 6 to 12 years who come with a history of a cold which has persisted from 10 days to 3 weeks with a cough especially at night, which does not respond to the various cough syrups, may be relieved of their cough within 48 hours after the injection of chloriodized oil. This preparation consists of 27% iodine and 7% chlorine in peanut oil. It does not destroy bacteria in vitro.

The mechanism of the action of chloriodized oil is still obscure, as is true of many very valuable therapeutic agents in medicine. Sulfanilamide does not kill bacteria in vitro, but in the presence of tissue it produces a compound which is bactericidal. Iodides

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are not germicidal but they may cause lysis and release bacteria as well as promote resolution of diseased tissues. Prontosil (4′-sulfamyl-2,4-diaminoazobenzene hydrochloride) is not active in vitro but is active in the presence of body tissues.

Suspensions of chloriodized oil could be inactive in a test tube, as dissemination is almost nil. However, I venture that low metabolic activity may give free iodine, as symptoms of iodism may and do occur after injection into an artrum.

Chloriodized oil is injected at intervals of 6 to 10 days, and often one injection is sufficient to clear the infection in a child. Infection of longer duration may require several injections. If the lesion is chronic, there should be no hesitation in making a large opening under the inferior turbinate. In all persistent infections in children, I find the use of thyroid and iodine of the greatest value.

In adults with frequent infections, recurring colds, and persistent bronchial cough, an infection of the antrum is to be suspected until there is proof otherwise. This holds true for corneal ulcers and persistent infection in an ear, which are usually accompanied by maxillary sinus infection.

Many patients come complaining of a persistent cold and bronchial cough which has extended over four to six weeks following an acute rhinitis. Various cough mixtures have been of no avail. It is surprising that these patients are often relieved within 48 hours after a single injection of chloriodized oil.[2] The relief is often phenomenal. It is astounding that a preparation which has no effect on bacteria in vitro should produce this astounding result familiar only to one who has had experience in the use of this preparation. Suprisingly, even chronic infections where operation has been refused, often obtain remarkable relief following interval injections.

I rarely, if ever, irrigate a maxillary sinus. Water mixed with infection and pus incubated at body temperature is to be avoided.

The one exception is the acute infection with foul-smelling pus following the extraction of an upper tooth. Here penicillin powder mixed in water in concentration is injected with a 5 cc. syringe of five consecutive days, then followed by chloriodized oil injection at four- to six-day intervals for several injections. I have had excellent results following this routine except where the lesion is an old chronic one requiring surgery.

Indication for Surgical Intervention

Any chronic infection of the maxillary sinus usually justifies a surgical procedure. This is particularly true where the infection is accompanied by corneal ulcers, lesions in the uveal tract, frequent and persistent bronchial cough, behavior problems in children,[3] and mental depression.

In chronic cases, a chloriodized oil injection will show an irregular scalloped outline in the filling defect. I prefer an intranasal antrotomy[4] in preference to a Caldwell-Luc operation. The surface area of the maxillary sinus is large, and, if completely denuded of its mucous membrane, it will never recover with a normal membrane. Often, repair is by a very thick fibrous tissue membrane with sparse cilia. Unless the opening under the inferior turbinate is large and remains widely opened, further trouble usually occurs. Thus, the success of the Caldwell-Luc operation depends more on the size and the position of the opening under the inferior turbinate than upon the complete removal of the sinus membrane.[4] It is important that the pars membrana of the middle meatus be retained, as here cilia are most active and an intact membrane prevents secretion from the frontal and the ethmoid cells from entering the maxillary cavity. Often an antrotomy is criticized because of a small opening or a complete closure following the procedure. It means that the antrotomy was improperly performed. Success depends upon a very definite precise procedure[5] to maintain a proper opening. It means that the opening

must be large and well rounded posteriorly using a downward biting Hajek-Koffler forceps pressing the nose well to the opposite side and working without fear of injury to the descending palatine artery. The steps of this operation and effect of compared instruments were carefully worked out on frozen sagittal head sections and presented by me some years ago.[4]

The antrum is packed with iodoform gauze, which may remain from 3 to 10 days. I advise removal if it becomes particularly uncomfortable to the patient. An applicator with a split tip is used in removing the packing.[5] No doubt the pressure of the packing helps in the recovery of the antrum membrane[6] or in the production of a new one. By means of a Pratt curette, all angles of a sinus can be reached[5] and the floor of an infraorbital ethmoid cell may be removed. Islands of membrane will remain which will serve in epitheliating the cavity. After-treatment consists in making applications of weak silver nitrate at intervals of a week or 10 days until the walls of the sinus feel smooth and dry. Very often little attention is required after the primary operation.

Frontal Sinus

In acute fulminating frontal sinusitis I advocate an incision through the periosteum in a line midway between the inner canthus and the midline of the nose, followed by the use of a common fingernail drill,[7] which is held at right angles to the medial wall of the orbit. Often fluid will be relieved under pressure. A few strands of absorbable surgical suture afford a good drain, and the cavity may be irrigated with an antibiotic for several days, by which time drainage has occurred through the nasal frontal duct and the skin is permitted to close.

In adult patients with an acute frontal sinus, after the shrinkage of the membrane and cocainization, the frontal may be gently entered with a small double curved frontal sinus cannula, followed by irrigation or suction. If the condition has existed for four or five days, as is often the case in patients who have had repeated attacks, I do not hesitate to fracture the middle turbinate immediately and cut away the anterior ethmoid cells with a Gruenwald forceps.[7] It will permit an easy entrance to the frontal sinus with a large cannula or a Pratt curette,[7] which consists of a small ring set at an angle of 45 degrees to the handle. The nasal frontal duct is never curetted or rasped, and never have I seen the duct closed by fibrous tissue following this procedure, properly performed.

Lateral roentgen views prior to operation[7] will tell one whether the internal approach is feasible. If not, then external drainage becomes imperative and should be done early to prevent intracranial complications. Should osteomyelitis occur requiring operation, the posterior wall of the frontal sinus should always be removed. Epidural or subdural abscesses occur early in the infection.

Intranasal approach to the frontal sinus has been most satisfactory in chronic frontal sinusitis, probably owing to the maintenance of a bony canal. In the external operation of the Lynch type this bony wall is sacrificed, and closure with interval infections occurs years later. In cases of secondary operations in the absence of a bony canal, I have found Ingals' gold tube[7] most helpful. This tube is to remain permanently.

If a primary radical frontal is performed, I prefer an incision just above the eyebrow. Sufficient bone is removed to expose and clean the sinus cavity. Ethmoid cells are removed by combined direct and intranasal approach, the bony canal of the nasofrontal duct being rigidly maintained.

Dr. Goodale[8] has recommended tantalum to prevent adhesions; he has used it with success in cases where the bony canal has been sacrificed.

Mucoceles

Muco- or pyroceles are lined with epithelium, and the primary operation should be intranasal, removing only the tip

of the lower portion of the mucocele. Thus will be maintained a nasofrontal duct lined with epithelium and a perfect skin graft.[9] The external deformity is corrected by external pressure on the thin overlying bone. The success of this operation depends upon the minimum of surgery and the advantage of the epithelial lining of the cavity which nature has prepared for us. Skin grafts have been advocated to maintain the nasofrontal passageway. Why destroy the one already awaiting us?

Ethmoidectomy

I prefer to save the middle turbinate if possible. If it is large and cystic it is split longitudinally by a sharp scalpel and the lateral half removed. The medial half is a protection to the cribriform plate and the olfactory area.

Gruenwald forceps are used to open the ethmoid labyrinth, and upward and downward biting Hajek-Koffler forceps are most helpful in biting clean and rounding out the anterior and posterior cells without injury to the lamina papyracea. A Pratt curette is a helpful accessory.

Sphenoidectomy

The membrane over the anterior wall is turned downward in an effort to avoid injury to the sphenopalatine artery, and the anterior bony wall is removed with a downward-biting Hajek-Koffler forceps. The anterior membrane is then pressed posteriorly and secured by iodoform packing, which is retained for five to seven days as a guard against bleeding following a possible injury to the sphenopalatine artery.

Finally, I would like to say a word in regard to the importance of chloriodized oil injections into the sphenoid sinuses in optic neuritis.[10] Opening of the sphenoid and the posterior ethmoid cells often gives immediate relief, but the same relief can be obtained by a simple injection of chloriodized oil into the sphenoid sinus on the affected side. I have ceased to do surgery in these cases since the oil injections have been equally satisfactory.

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Intranasal Chromophobe Adenocarcinoma

Report of a Case

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Chromophobe adenoma is a common intracranial tumor of adults. It usually arises in the vicinity of the pituitary stalk on the periphery of the gland where the chromophobe cells usually predominate. It is a globular encapsulated lesion that grows to large proportions and may be either solid or cystic.[1] The tumor causes deformity and destruction of the sella and sometimes gives rise to symptoms of hypopituitarism. It causes compression of the optic chiasm and frequently produces characteristic bitemporal hemianopic visual field defects. Microscopically the tumor consists of polygonal cells arranged in columns, or alveoli about a stroma of branching sinuses. It is separated by a capsule of connective tissue from the normal gland. Under high magnification the uniform cells reveal an angular cytoplasm and round to oval hyperchromatic nuclei with little evidence of mitosis.

These tumors may extend above the tentorium [2] to compress and distort the optic nerves, chiasm, or tracts. Photophobia and blurred vision may precede the visual field defects, which are usually variations of bitemporal hemianopsia. As time goes on, the growth of the tumor becomes eccentric, growing predominantly to one side with loss of vision in the ipsilateral eye. There are other symptoms which may precede visual field changes. These arise from pressure on adjacent oculomotor or other cranial nerves or the adjacent incus. Headache is the first symptom in about one-third and is present in at least three-fourths of all patients. It is usually frontal and often intense behind the eyes or in the temples. Optic atrophy of the primary type due to pressure on the optic nerves is present in a large percentage of patients when they first present themselves for examination.[3] It is interesting to note that the tumor must rise 10 to 30 mm. above the sella to compress the optic nerves or chiasm. Optic atrophy is usually sooner or later bilateral but may remain unilateral. There are no choked disks.

In review, the usual features of chromophobe adenoma are the following: optic atrophy, bitemporal hemianopsia, deformity of the sella turcica, headache, occasional oculomotor or other localizing symptoms. Added to these symptoms sometimes are endocrine disturbances such as amenorrhea, loss of libido, increased weight, change in skin or hair, high carbohydrate tolerance, and decreased basal metabolism. In a study of urinary hormone excretion female sex hormone may be absent, 17-ketosteroids low or absent, corticoids very low, and estrogens absent.[8] Adenomas recur but do not metastasize. They have been stated to be radiosensitive, but further accumulated statistical evidence on longer follow-ups is necessary to justify that conclusion.

Chromophobe adenocarcinoma in both local and general symptoms produces a similar picture.[4] It is highly malignant. It develops more quickly than adenoma. It seldom if ever metastasizes. The pressure of the space-taking lesion causes the dura covering the sella to yield; then the bone of the sella perforates, enlarging in the typical manner extending down into the sphenoid sinus covered by a thin shell of bone. The tumor may appear in the nasopharynx, project anterior, posterior, or lateral to the chiasm.

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or insinuate itself between the dura and the base of the skull, spreading into the temporal fossae.[6] It usually remains globular, deeply indenting the diencephalon anterior to the optic chiasm.

Bailey and Cutler in 1940 [5] recorded one of the first examples of malignant chromophobe adenocarcinoma that invaded the nasal cavity. Malignancy was based on cell type and clinical behavior. A 31-year-old woman patient had an invasion of the base of the skull, sphenoid sinus, and right nasal cavity. At autopsy all tumor was directly continuous, and there were no metastases. Shea [9] in a paper on "Nasal Tumors of Pituitary Origin" quotes a study by Henderson in which 338 cases in Cushing's series yielded 8 extensions into the nasopharynx but none entered the nasal cavity directly.

Kay, Lees, and Stout [7] studied 192 cases of chromophobe adenoma operated on at the Neurological Institute and the Presbyterian Hospital, finding only 3 with nasal extension. Although continuity was not demonstrated, it was their opinion that the nasal growths represented direct extensions from the primary growths in the pituitary gland. While some chromophobe adenomas are called carcinomas, Bailey and Cutler do not believe that they ever metastasize. Whether the growths should be called malignant is of little importance. The significant fact is that the growths reached the nasal cavities directly and caused nasal obstruction. The cell structure and the rapid clinical course give origin to the classification of malignancy. The tumors do not split capsules and rarely if ever metastasize. Although invasion of the nasopharynx is not particularly infrequent, direct invasion of the nasal cavity as here reported, with clear-cut complete clinical record, with roentgen studies, histologic verification intracranially and intranasally, is rare.

Report of Case

A woman, aged 52, weight 100 lb., came for examination because of severe frontal headaches. These had recurred at intervals for two years. There was a history of allergy studies followed by hyposensitization therapy. The headaches were frequently accompanied by blurred right vision such as is not uncommon in hemicrania. In May, 1954, headaches located over the frontal region and deep in the head became severer. The attacks were often worse at 4 a. m., lasted one to two days and occurred once or twice weekly, with freedom from headache in the intervals. A medical consultant prescribed liver therapy for mild anemia and thyroid therapy for mild hypothyroidism. In March, 1955, the headaches seemed aggravated by emotional stress resulting from the sudden death of her only brother and increasing responsibility in the care of a senile confused father with cancer of the colon. In September, 1955, the headaches were occasionally accompanied by nausea and vomiting, for which her physician prescribed sedatives and chlorpromazine (Thorazine). A careful otolaryngological examination on Oct. 1, 1955, revealed no abnormalities other than typical allergic nasal membranes. Ophthalmological findings were normal, with vision 20/20 in each eye; fields, fundi, and tension normal. On Oct. 31, 1955, diplopia suddenly developed. A neurosurgeon was consulted, and the patient was admitted to the Evanston Hospital for study. Ptosis developed in the right eye on the evening of admission. The ophthalmological report the following day was as follows: vision right eye 20/40, left 20/20, fields, fundi, and tension normal, and no papilledema. There was involvement of the right oculomotor and trochlear nerves. On Nov. 9, 1955, x-ray examination of the skull revealed rather

Fig. 1—X-ray of base of skull revealing tumor tissue replacement of bone in the region of the pituitary and body of the sphenoid bone. Burr holes are noted from the transfrontal craniotomy. (June 6, 1957.)

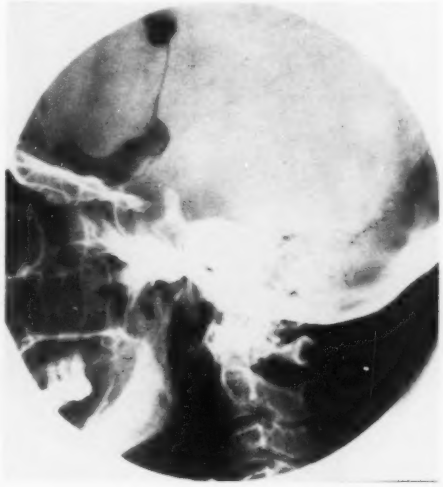


Fig. 2.—X-ray of the skull revealing advanced bone absorption in the base of the skull and walls of the right orbit. Burr holes are present from the transfrontal craniotomy. (Nov. 27, 1957.)

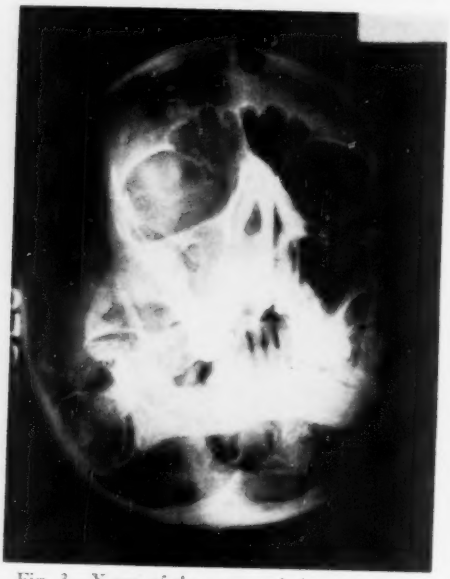
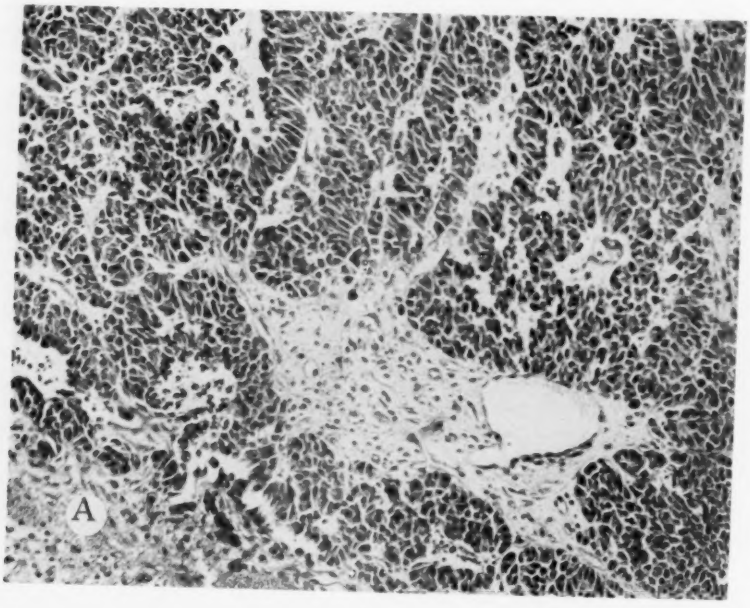


Fig. 3.—X-ray of the paranasal sinuses revealing destruction by the tumor of the median and inferior right orbital walls with invasion of the right orbit, ethmoid, antrum, and nasal regions. (Nov. 27, 1957.)

Fig. 4A.—Section (×145) revealing highly cellular, dark-staining chromophobe adenocarcinoma nuclei.



marked demineralization of the posterior clinoid processes. The floor of the sella turcica appeared to have been eroded and the erosion extended into the roof of the sphenoid sinus. This was diagnosed a space-occupying lesion, probably pituitary adenoma. Paranasal sinuses were normal. On Nov. 11, 1955, a right cerebral arteriogram was made after instillation of 35% iodopyracet (Diodrast) compound in the right common carotid artery. An excellent demonstration of normal intracranial circulation was accomplished in frontal and lateral positions.

On Nov. 15, 1955, under thiopental (Pentothal sodium), nitrous oxide, and oxygen anesthesia, a transfrontal craniotomy was done (Dr. Joseph Tarkington). The surface of the cortex was normal. The sella surface had a small 6 mm. tumor mass protruding. With rongeur forceps, the sella was opened and several small masses of tumor removed. The cavity was filled with absorbable gelatin sponge (Gelfoam) and closure made with drainage.

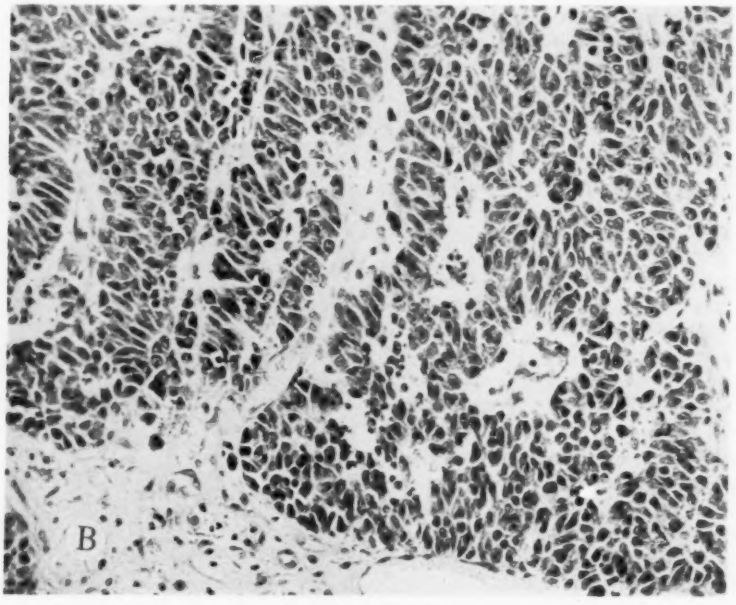
Dr. Henry H. Teloh, Evanston Hospital Pathologist, reported the following: "There are present several clumps or masses of dark cells which are arranged in cords and have some papillary pattern. These cells are polyhedral and have markedly hyperchromatic basophilic nuclei with many mitotic figures. There is slight variation in the size and shape of the neoplastic cells and the presence of occasional unusually dark large nuclei. The stroma has abundant blood vessels. Diagnosis: Chromophobe Adenoma."

Two weeks postoperatively, Dr. Anna Hamen, Evanston Hospital radiotherapist, started deep x-ray therapy. A skull pituitary area 2×4 cm. was plotted. There were 55 rotations with a total of 278 r minimum dose on the surface in any area and 834 r at the depth of the lesion, a total of 25 treatments in 33 days from Nov. 9 to Dec. 31, 1955, a total of 4170 r.

On Dec. 7, 1955, the record notes normal fundi, normal visual fields, decreasing right ptosis, and ocular motility good except for covergence.

There were no important new findings for 16½ months. On May 15, 1957, examination revealed slight proptosis of the right eye, right vision 20/100, and slight exotropia. One month later, on June 15, 1957, the right eye was blind, but the left, including the field, normal. The right nostril was filled with tumor mass presumed by the patient to be an allergic reaction. On July 11, 1957, the patient was taken to the operating room for a biopsy specimen. Since the tumor did not seem dangerously vascular, all tumor tissue in the right nostril and right ethmoid region was removed. A probe passed easily into the orbit to the globe. The tentative diagnosis was intranasal extension of chromophobe adenoma previously operated on by intracranial approach. The tissue description by Dr. Teloh was as follows: "Grossly the specimen consists of 5 grayish white soft to

Fig. 4B.—Section (× 250) revealing cell structure of chromophobe adenocarcinoma.



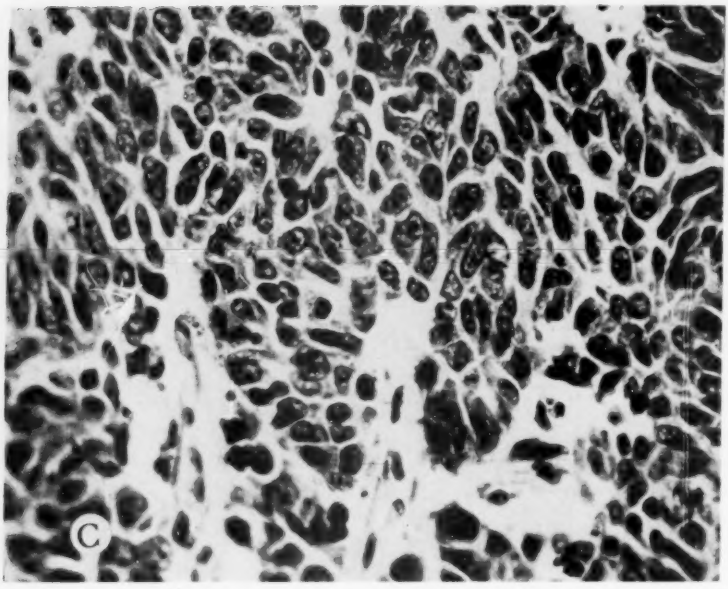


Fig. 4C.—Section (× 475) revealing mitotic activity in nuclei of chromophobe adenocarcinoma.

firm masses of tissue. Microscopically sections reveal a tumor which is composed of sheets and masses of moderately to markedly pleomorphic dark epithelial cells. They have many mitoses and frequent necrotic areas. The diagnosis: Chromophobe adenocarcinoma of the nasal cavity."

After surgical consultation it was decided that radical surgery was injudicious because of the extensive involvement of the base of the skull. Two weeks postoperatively, Dr. Hamen started further deep x-ray therapy over the nose, left lateral skull, and forehead. From July 22, 1957, to Aug. 13, 1957—22 days—3423 r was given, a total for the two series of 7593 r. Chest films were normal.

Two months after this series of x-ray treatments the left eye became blind. The patient was totally blind for the final three months of life but remained mentally clear until going into a terminal 24-hour coma. Death was apparently due to gradually increasing pressure on the vital centers produced by the tumor growth, and occurred five months after the termination of the second series of x-ray treatments.

Continuous tumor tissue, histologically identified as chromophobe adenocarcinoma, extended from the pituitary region of the base of the skull through the right orbit, replacing its inferior and medial walls and extending into and filling the right antrum and right nostril.

Summary and Conclusions

The term chromophobe adenocarcinoma seems justified from the histologic picture of active mitoses and clinically by the rapid development.

No metastases were noted in the cases reviewed or in the case presented.

Fig. 5.—Terminal picture showing the external involvement of the right orbit and nostril.

Roentgen therapy used to safe maximum dosage was of value apparently only in retarding growth.

Chromophobe adenocarcinoma directly invading the nasal cavity is apparently rare, since Cushing's large series yielded no direct nasal invasions, and the series studied by Kay, Lees, and Stout only three cases.

Stereo studies of the skull should periodically be made for persistent, unusual, and localizing headache symptoms.

636 Church St.

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Associated Carcinoma of Larynx and Lung

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The association of carcinomas of the larynx and lung is illustrated by two patients who have come to our attention. In the first patient the association was concurrent; in the second it was consecutive.

Lewis and Schaff [1] reported 12 patients with this combination. Cahan [2] was able to collect 19 examples from Memorial Hospital, New York, in a survey of 2052 cases of lung cancer that covered a 28-year period. He found 18 examples of associated carcinoma of the head and neck other than laryngeal in the same series of lung cancer cases. Thomson and Schaff [3] reported an amazingly high incidence of associated lung and laryngeal cancer; 5 out of 20 patients coming to postmortem examination with laryngeal cancer were shown to have developed concurrent or subsequent bronchial carcinomas.

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Report of Cases

CASE 1.—A white man, age 66, was treated in 1944 for carcinoma of the vocal cords by means of irradiation following removal of the thyroid cartilages. The microscopic diagnosis was epidermoid carcinoma, Grade III. In 1951, the patient suddenly developed symptoms resembling bronchial asthma, and in 1952 an opacity was seen in the right lower lung field on x-ray examination. Bronchoscopy and cell study were negative, but thoracotomy revealed a smooth, well-outlined mass near the lateral aspect of the hilus of the middle lobe. A right middle lobectomy was performed, the microscopic diagnosis being epidermoid carcinoma. The patient remained

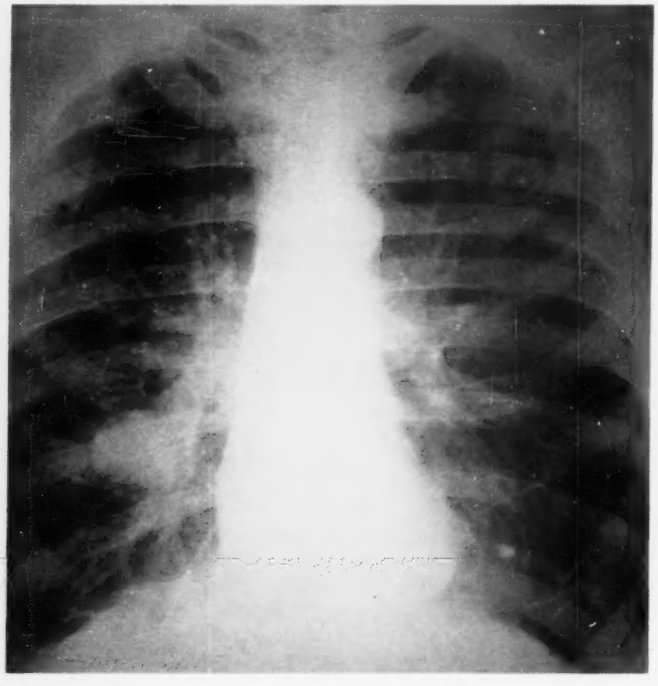

Fig. 1 (Case 1).—Epidermoid carcinoma of lung.

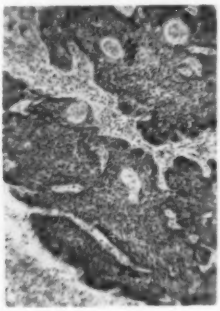

Fig. 2 (Case 1).—Carcinoma of larynx, 1943 (low power).

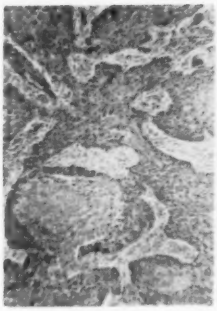

Fig. 3 (Case 1).—Carcinoma of bronchus, 1952 (low power).

alive for one year, then died suddenly of myocardial infarction, there being no apparent recurrence of his carcinoma (no autopsy).

Comment: This case illustrates the separate occurrence in time (metachronous) of these two lesions, and the successful treatment of the laryngeal lesion by a now outmoded method.

CASE 2.—A Negro man, age 62, was first seen on March 16, 1957, complaining of hoarseness for three weeks or longer, and of some soreness of the throat. The patient also complained of a constricting retrosternal feeling on exertion. The larynx appeared inflamed and the cords polypoid on mirror examination. After one week without smoking cigarettes, the inflammation of the larynx was less and the cords had a ragged appearance. Direct

Fig. 4 (Case 2).—Carcinoma of larynx, 1957 (low power).

Fig. 5 (Case 2).—Carcinoma of bronchus, 1957 (low power).

laryngoscopy and biopsy on March 28, 1957, revealed Grade II to III epidermoid carcinoma from the left vocal cord. The EKG showed severe myocardial damage and complete heart block, and chest x-ray showed enlargement of the left ventricle and enlarged right hilar shadow. Laryngoscopy was therefore again performed, and also bronchoscopy. Biopsy of the right vocal cord was reported as chronic inflammation. The entire intrinsic larynx appeared polypoid, and the ragged lesion of the left cord extended subglottically and clinically also involved the anterior half of the right vocal cord. Bronchoscopy revealed a circumscribed, elevated, reddish lesion at the orifice of the right upper lobe bronchus, appearing about 1 cm. in diameter. Biopsy of this lesion was also reported epidermoid carcinoma of bronchus, Grade III.

In spite of the patient's obviously poor cardiac status, it was decided to treat his cancers vigorously because they would otherwise surely kill him. It was felt that treatment of the larynx should be deferred until the resectability of the lung was determined. Preliminary tracheostomy was performed on April 10, 1957. On April 23, 1957, right thoracotomy was performed. A portion of the surgeon's report is as follows: "Palpation of the lung revealed no evidence of a tumor mass in either of the three lobes. The bronchi and the trachea were exposed posteriorly, but a mass could not be palpated. . . . To facilitate dissection, the upper lobe bronchus was divided so that the lung could be removed and later with ease dissection carried back to the carina. The middle and lower lobe branches of the pulmonary artery were mobilized and divided between ligatures." Within five minutes, cardiac arrest occurred, and despite cardiac massage and defibrillation through the open pericardium, the patient died.

Comment: This second case illustrates the simultaneous (synchronous) appearance of a laryngeal and bronchial carcinoma. It is chiefly remarkable in that it appears that the bronchial lesion was discovered almost by accident, since it is difficult to believe the small lesion later visualized could have caused any demonstrable x-ray changes. The pathologist described the bronchial lesion as being only 1 cm. across, and stated it had not invaded as far as the bronchial cartilage,

so that it could be described as being just beyond an "in situ" carcinoma.

General Comment

Although Cahan and his associates were concerned over the difficulty of determining whether the lung lesion could be established as primary or metastatic, most writers agree that when a laryngeal carcinoma occurs prior to, or is concomitant with, a tumor in the lung the pulmonary lesion should be considered as a second and independent primary. The following reasons are given:

1. The time interval in many of the cases reported is sufficiently long to make metastases unlikely.

2. Laryngeal carcinomas rarely metastasize to the lungs without first developing widespread involvement of the cervical lymph nodes.

3. The laryngeal and bronchial carcinomas are frequently different histologically.

4. It is unlikely that cells from the laryngeal carcinoma would make their way into the bronchi because the flow of respiratory secretion is in the opposite direction.

5. Bronchial carcinomas almost never precede laryngeal carcinomas in point of time.

6. Metastatic carcinoma to the larynx is rare and no cases of bronchial carcinomas metastasizing to the larynx have been reported.

Multiple primary neoplasia is by no means rare. Actually, the person who has had one cancer is more likely to develop a second independent cancer than another person is to develop his first. Warren and Gates [4] consider the tendency for a person to develop a second cancer to be 11 times greater than that of the patient without a previous cancer.

Billroth, in the 18th century, called attention to the occurrence of multiple primary cancers. The subject has been reviewed a number of times.[4-7]

Warren and Gates found 194 instances of multiple primary malignancy in a series of 2829 postmortem examinations of cancer patients. This represents an incidence of 6.8%. They compiled a 3.9% average for the United States, but this is probably less than the actual incidence. Slaughter [5] found that multiple primary malignancies represented 3.9% of all cases of carcinoma in the records he reviewed.

There is a tendency for multiple primary cancers to occur in the same organ or in paired organs. Most multiple lesions occur in the skin and in combination with the skin. The gastrointestinal system is next most commonly affected, and this is followed by the genitourinary system. The highest incidence in paired organs in order is in breasts, ovaries, testicles, and kidneys. Multiple primary lesions involving two separate systems occur most frequently in the gastrointestinal and genitourinary systems. Second in frequency are combinations of breast and genitourinary system.

The time intervals recorded in the literature vary from 0 to 14 years, with an average of 3.2 years. The Memorial Hospital, New York, group distinguish synchronous and metachronous lesions. When the interval between the diagnosis of first and second tumors is less than six months, the lesions are considered synchronous. When the interval is greater than six months they are considered metachronous.[4]

Treatment

When two carcinomas are discovered simultaneously, the more lethal lesion should be attacked first.[2] In the lung-larynx combination, the more lethal lesion is the bronchogenic. It would be unwise to subject a patient to laryngectomy only to find later that his bronchial carcinoma is not resectable. It is good practice to perform preliminary tracheostomy before thoracotomy in order to avoid the necessity of passing an endotracheal tube through the malignant glottis and to eliminate the danger of postoperative laryngeal edema. Tracheobronchial toilet is facilitated by tracheostomy in patients who are subjected to thoracotomy. If the patient is found to have a resectable lung lesion, definitive laryngeal surgery can be carried out after a reasonable period of convalescence. Otherwise palliative irradiation may be used for the larynx.

Summary and Conclusions

Case reports of two patients with associated carcinoma of the larynx and bronchus are presented. A brief discussion of the subject of multiple primary neoplasia with particular reference to the respiratory tract emphasizes that patients who have carcinoma of the larynx should be observed carefully for the appearance of carcinoma of the lung. These cases probably always represent multiple primary lesions, and the association of carcinoma of the bronchus and larynx in the same patient is apparently fairly frequent. Unless the combination of multiple primary neoplasia is borne in mind, the treated cancer patient may be denied aggressive attack on a second and independent malignant lesion.

Dr. Henry W. Maxfield, of Mason City, Ill., and Dr. Robert T. Patey, of Springfield, Ill., gave us permission to present the two illustrative cases.

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Cerebral Mucormycosis

A Report of Three Cases

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A fulminating infection of the nose, sinuses, orbits, and cranial structures may be caused by a fungus of the Mucorales order, and may closely resemble acute sinusitis of bacterial or viral origin. The disease has occurred with increasing frequency in the past 10 years and will undoubtedly become more important with wider use of antibiotics, corticosteroids, and other chemotherapeutic agents.

Certain fungi are remarkable in their ability to produce disease in the absence of underlying illness (blastomycosis, coccidioidomycosis, etc.), while others usually complicate a preexisting infection (Monilia, Aspergillus, etc.).[27] Most of the diseases they produce are nonfatal, but two notable exceptions are blastomycosis and mucormycosis. Either of these infections may prove fatal, but of the two mucormycosis is more serious, sometimes killing the patient in a matter of days. The causative organism becomes invasive and nearly always lethal in patients being treated with chemical agents such as corticosteroids, antileukemic preparations, and especially antibiotics when given to an uncontrolled diabetic.

Historical

The first indication of the pathogenicity of the Mucorales came from Lichtheim's[4] experimental studies on rabbits in 1884. In 1885 Paltauf[5] first described a patient having a systemic fungus infection involving the central nervous system. The fungus was identified as belonging to the order of Mucorales, and he coined the term "mycosis mucorina." In 1943, 58 years after the first case of cerebral mucormycosis was reported, Gregory et al.[6] stimulated interest in this group of fungi by an extensive review article which included three case reports. The next reported case[7] was in 1947, and since that time no less than 10 articles containing case reports have been published.[2,8-16] The case reported by Harris[2] was of great significance, for all 13 previously reported cases had been fatal, whereas his was the first case to survive the disease and the only one to have been diagnosed while still alive. A second case to survive the disease was mentioned by Baker[16] and is soon to be published.[1] As can be seen from the Table, diabetes was the concurrent disease in 15 of the 21 listed cases. The youngest recorded case was a 20-day-old girl, and the oldest was a 75-year-old man. Two cases reported to have survived the disease, 14 and 17, were Negroes, and both had uncontrolled diabetes as the concurrent disease.

Etiologic Agent

The etiologic agent in cerebral mucormycosis is a true fungus. The true fungi are divided into two classes on the basis of their hyphae structure, one class being septate, the other nonseptate. To the class with nonseptate mycelia (Phycomycetes) belongs the family Mucoraceae. Mucor and Rhizopus are representative genera which can be identified only by their characteristic sporulation. Characteristic sporulation is usually not present in the tissue biopsy specimen, so diagnosis usually requires culture. Species within each genus are

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Summary of Reported Cases of Cerebral Mucormycosis

Case No.	Reported By	Age & Sex	Concurrent Disease	Organs Involved	Vascular Lesions	Orbital Signs
1	Paltauf,[5] 1885	Adult M.	Intestinal ulceration	Brain, lungs, intestine	Not known	Not known
2	Gregory et al.,[6] 1943	43 F.	Diabetes	Orbit, brain, meninges, pituitary, cavernous sinus, internal carotid	Thrombosis of left cavernous sinus, left internal carotid, and circle of Willis	Severe, left eye necrotic
3	Gregory et al.,[6] 1943	52 F.	Diabetes	Orbit, ethmoid air cells, brain	Thrombosis of orbital and cerebral vessels	Severe, right eye enucleated
4	Gregory et al.,[6] 1943	75 M.	Diabetes	Orbit, brain, meninges, spinal cord, pituitary	Invasion of meningeal vessels	Severe, right eye proptosed, bullous edema and congestion of conjunctiva
5	Le Compte et al.,[7] 1947	57 M.	Diabetes & hemochromatosis	Orbit, brain	Invasion of cerebral vessels	Severe, right eye inflamed, swollen and purulent
6	Baker & Severance,[8a] 1948	3 F.	Diabetes	Lungs, lymph nodes, nerves, orbit, internal carotid, brain	Thrombosis of left internal carotid, orbital and cerebral vessels	Left pupil larger than right, hemorrhage in left eye
7	Wolf & Cowen,[9] 1949	42 M.	Diabetes	Brain, cavernous sinus	Thrombosis of cavernous sinus	Not described
8	Stratemeier,[9] 1950	32 F.	Diabetes	Brain	Invasion of cerebral vessels	Left eye swollen and ptosed
9	Kurrein,[10] 1954	5 mo. M.	Acute nephritis, uremia, antibiotics	Brain	Invasion of cerebral vessels	None
10	Martin et al.,[11] 1954	2 1/2 mo. M.	Diarrhea, antibiotics	Orbit, meninges, face, internal carotid, esophagus, pleura, lungs, pancreas, bone marrow	Thrombosis of left internal carotid	Severe left orbital necrosis
11	Bauer et al.,[12] 1955	40 M.	Diabetes	Orbit, nasal sinuses, brain, internal carotid	Thrombosis of right internal carotid	Complete right ophthalmoplegia
12	Bauer et al.,[12] 1955	25 F.	Diabetes	Orbit, brain	Invasion of cerebral and meningeal vessels	Periorbital edema, fixed pupil, and retinal edema
13	Gunson & Bowden,[13] 1955	5 F.	Chronic glomerulonephritis	Brain, internal carotid	Thrombosis of left internal carotid	None
14	Harris,[9] 1955	14 F.	Diabetes	Turbinates, palate, tongue orbit, cranial nerves	Not known	Severe, complete ophthalmoplegia with blindness
15	Foushee,[14] 1956	65 F.	? Diabetes, antibiotics, postcholecystectomy	Left olfactory bulb, orbit, meninges, brain	Thrombosis of left internal carotid	Edema, complete ophthalmoplegia, fixed dilated pupil, 5th & 7th nerve function lost
16	Jackson et al.,[15] 1957	20 days F.	Congenital heart disease, pneumonia, antibiotics	Brain, intestine	Invasion of cerebral vessels	Transient edema of eyelids
17	Baker et al.,[16,18] 1957	Adult	Diabetes	Ethmoid sinus, orbit	Not known	Marked periorbital swelling
18	Baker,[16] 1957	62 M.	Cirrhosis	Sinuses, left internal carotid meninges, brain	Thrombosis of left internal carotid	Chemosis and proptosis

19	Case 1		72 M.	Diabetes, antibiotics	Turbinates, external nose, right internal carotid, pituitary, meninges, brain, orbit, sinuses	Thrombosis of right internal carotid orbital and cerebral vessels, sphenopalatine artery	Severe, complete right ophthalmoplegia with blindness, 5 & 7 CN function lost
20	Case 2		6½ F.	Diabetes, antibiotics	Turbinates, orbit, sinuses, brain, meninges, internal carotid, pituitary	Thrombosis of left internal carotid, cerebral and meningeal vessels, sphenopalatine artery	Severe, complete left ophthalmoplegia with blindness, 5 & 7 CN function lost
21	Case 3		50 F.	Diabetes, antibiotics	Turbinates, maxillary sinuses, maxillary bone, alveolar ridge, orbit	Right infraorbital artery, right sphenopalatine artery	Severe, complete right ophthalmoplegia with blindness, 5 & 7 CN function lost

distinguished by their sporangia, sporangiospores, and zygospores. In contrast to Mucor, the genus Rhizopus is characterized by groups (fascicles) of unbranched sporangiospores which arise from nodes of runners (stolons) opposite root-like hyphae or rhizoids. According to Castellani,[17] the family Mucoraceae are very abundant in nature, where they exist largely as saprophytes in the soil, in manure, on fruits, and starchy foods such as bread. They have been cultured from the nose and gastrointestinal tract of normal individuals. Garrett[18] states that a fast rate of mycelial growth and rapid spore germination enables these fungi to progress rapidly whenever a suitable substrate is encountered. Under aerobic conditions and in the presence of a sugar-containing substrate, such as Sabouraud's glucose agar, the fungus readily sporulates, filling the Petri dish in five days. Baker[19] demonstrated the hyphae with ordinary hematoxylin and eosin staining and even better by hematoxylin staining alone. He noted that hyphae tend to be very thick where conditions are favorable to growth, quite narrow in infarcted or necrotic tissue.

Pathology

Definite tissue invasion by the organism must be found on histologic examination if it is to be considered pathogenic. The hyphae show a striking affinity for arteries, penetrating their walls and growing into their lumens to produce thrombosis (Fig. 3).

Fig. 1 (Case 1).—Note black necrotic area of infraction and periorbital swelling with discoloration.

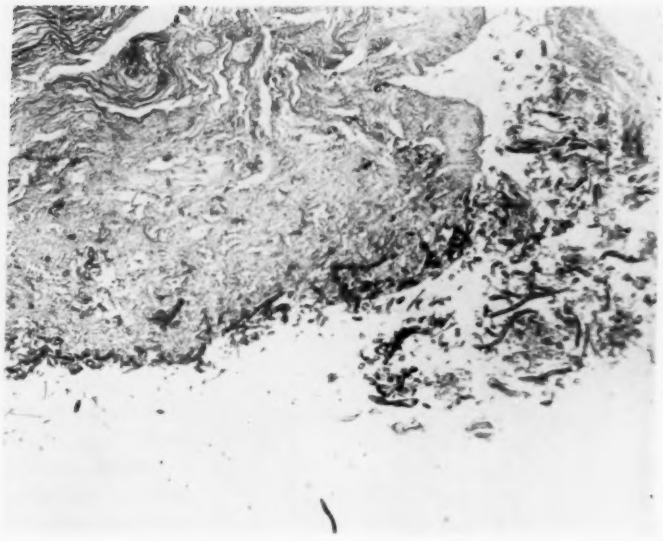


Fig. 2 (Case 1).—Section of nasal lesion showing hyphae invading tissue.

The lymphatics, veins, and nerves are invaded later. There is remarkably little inflammatory reaction to the fungus, whether isolated or in clusters.[12] What little inflammation does occur is found in tissues injured by vascular obstruction. Infarction is characteristic of the disease. (Fig. 2).

In marked contrast to bacterial infections of the paranasal areas the cavernous sinus usually escapes thrombosis, but the internal carotid artery passing through its lateral wall is plugged with hyphae (Fig. 6). In prolonged cases of cerebral mucormycosis where the disease was well established, hyphae have been found disseminated throughout many of the body organs.

There are several remarkable instances of the unexpected discovery of this fungus on routine examination of tissues from patients who recovered. Ash and Raum [25] presented

Fig. 3 (Case 1).—Hyphae seen invading wall of internal carotid artery.

Fig. 4 (Case 2).—Autopsy specimen showing black thrombosed turbinate.

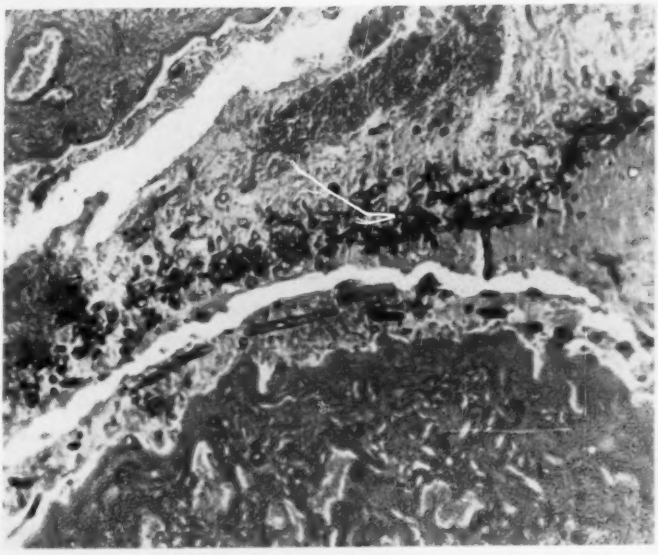


Fig. 5 (Case 2).—Section of thromosed turbinate showing active invasion by hyphae.

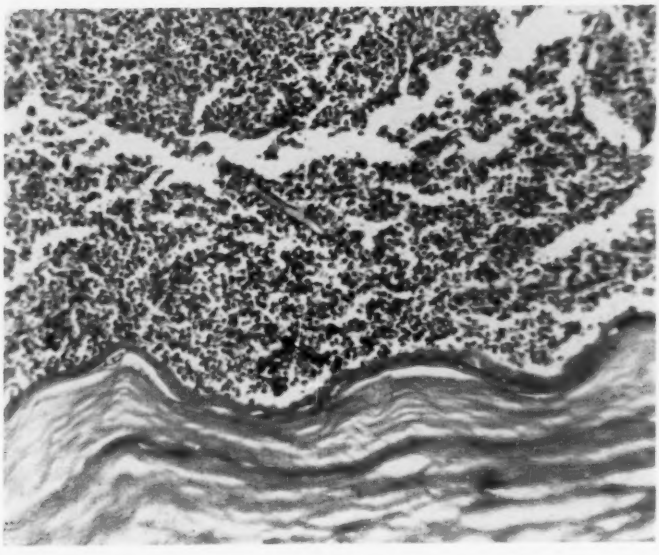


Fig. 6 (Case 2).—Mycelia present in lumen of thrombosed internal carotid artery.

sections of a granulomatous nasal lesion demonstrating broad nonseptate hyphae invading the tissue. Wadsworth[26] found broad nonseptate hyphae invading the fundus of an eye which was removed because of Coats's disease. Veirs and Davis[30] demonstrated nonseptate hyphae invading the cornea after an injury was treated for several weeks with antibiotics and corticosteroids. These cases lend further authority to the opinion that the fungus becomes fatally invasive only when the patient's general resistance has been altered by metabolic disorders or chemical agents.

Experimental

Early experimental work on the pathogenicity of the Mucorales was reported by Lichtheim.[4] Baker and his associates[3,21,24,28,29] have studied pathologic reactions in experimental animals. Bauer et al.[20] produced fulminating cerebral mucormycosis in alloxan-induced diabetes in rabbits by intranasal instillation of a saline suspension of fungus spores and showed that the infection spread rapidly from the nasal mucosa to the adjacent tissues. The lesions produced closely resembled those occurring in man. In sharp contrast to the diabetic animals the nondiabetic controls showed an acute inflammatory reaction to the spores in the ulcerated nasal mucosa and absence of hyphae in the tissues. Corticosteroids favor dissemination of the organism in animal experiments,[21] possibly as a result of their diabetogenic effect, as suggested by Stefanini and Allegra.[22]

Report of Cases

Case 1.—A 72-year-old partially blind white diabetic man was admitted to the hospital because of swelling and redness of the right periorbital area.

One month before admission the patient experienced an episode of right facial pain which came on suddenly. The pain gradually subsided in a few days without treatment, leaving the right frontal sinus area sensitive to pressure. Two weeks prior to admission redness and swelling, without pain or fever, developed around the right eye.

This man was a known diabetic for over 20 years and was well regulated on 20-30 units of isophane insulin (NPH) daily. Sight in the right eye had been absent for two years and markedly diminished in the left eye for six months. There was a questionable history of sinus trouble for about 20 years, which never required treatment. The right leg had been amputated 18 months ago and the left leg amputated 6 months ago.

Admission physical examination revealed a well-oriented elderly thin white man. The temperature was 100.4 F, the pulse 64, respiration 20, blood

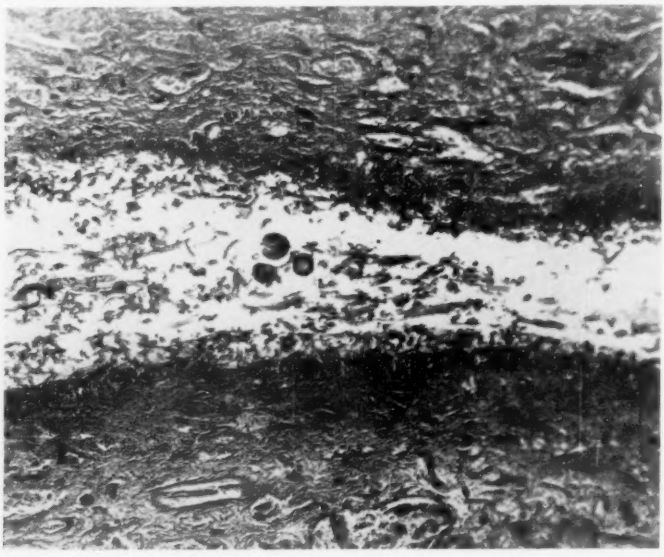


Fig. 7 (Case 2).—The aerial or reproductive mycelia are seen growing in the space between the turbinates.

pressure 130/76. The right periorbital tissues were discolored and swollen, with complete closure of the eyelids. The right frontal, ethmoid, and maxillary sinuses were tender to palpation and percussion. The right eye was proptosed and fixed but without palpable increase in intraocular pressure. The right cornea was cloudy, and the pupil small and fixed. Hemorrhagic dry adherent crusts were present in the vestibule of the right nostril.

The urinalysis revealed 3+ albumin, no sugar, and no acetone. The sediment was loaded with WBC and bacteria. The hemoglobin was 12 gm., WBC 37,600, with 70% segmented neutrophils. X-rays of the sinuses showed a pansinusitis, severer on the right, without fluid levels.

On the evening of admission aqueous and procaine penicillin were started, nasal vasoconstrictor drops were instilled hourly, and warm saline soaks applied to the right eye. Seven hours later the temperature rose to 102.5 F. At this time streptomycin was added to the antibiotic program. During the next day the patient remained well oriented, his temperature 102 F, and the urine free of sugar and acetone.

During the morning of the third hospital day the temperature was normal but by afternoon it had risen to 102.5 F. At that time an abscess was observed over the right nasolabial fold, with associated swelling of the adjacent lip and infraorbital area. The hemorrhagic dry crust in the right nasal vestibule seen on admission was found to be an area of dry black gangrene of the lateral nasal wall. The right maxillary antrum was irrigated with saline, with clear returns. A culture of the nasolabial abscess grew out hemolytic Staphylococcus aureus. The dosage of penicillin was increased and erythromycin added.

By the fourth hospital day the patient had become listless, lethargic, and incontinent. The right eye globe had become palpably soft. Blood chemistries at this time revealed a FBS of 266 mg. % and a CO_2 of 17.8 mEq. per liter. The area of gangrene in the right nasal vestibule had enlarged toward the right eye (Fig. 1).

The temperature remained elevated on the fifth hospital day and the patient became more lethargic. The area of gangrene had spread up to the inner canthus of the right eye. Parenteral bacitracin and cortisone were added to the therapeutic program without noticeable effect.

On the sixth and last hospital day the patient remained unresponsive. Several hours before death the right side of the face was observed to be paralyzed and the corneal reflex absent.

Examination at autopsy showed a large dark reddish-brown crusted gangrenous abscess of the right ala nasi and adjacent nasolabial soft tissues. The skin, soft tissues, and underlying mucous membrane were involved, with widespread cellulitis of the periorbital tissues on the right without frank ulceration. There was a blackish brown discoloration of the turbinates. Microscopic examination of these tissues revealed branching nonseptate hyphae.

The cavernous sinus was not thrombosed. There was a plug of homogeneous yellow material in the right internal carotid artery as it entered the cavernous sinus. A thrombus was seen in the right ophthalmic vein. Microscopic examination of the right internal carotid showed the plug to be composed of a mass of small cells having small teardrop dark-staining nuclei. The artery wall contained many large branching hyphae (Fig. 3).

The pituitary appeared grossly normal, but microscopic sections showed many large branching hyphae in the lumen and walls of small blood vessels.

The brain appeared moderately edematous, and the meninges over the pons showed a small amount of thick creamy purulent exudate. Microscopic sections revealed an acute basilar meningitis and thrombosis of the superficial layers of the pons, cerebellum, and frontal cortex. Many small abscesses with moderate to marked proliferation of microglia in the surrounding parencyhma were seen. In these regions the meninges and the meningeal vessels contained many branching hyphae.

Case 2.—A 6½-year-old white girl was brought to the hospital because of lethargy, deep rapid breathing, polyuria, and polydyspia.

The present illness began four months prior to admission with an episode of tonsillitis, which responded well to antibiotic therapy.

Two months later the mother noticed that her child had developed weight loss, polyuria, and polydyspia.

One month prior to admission the child had another attack of tonsillitis, which was treated with good results by intramuscular penicillin.

Four days prior to admission, the attending physician noted that the child looked ill, although her tonsillitis had cleared and her appetite was good.

Two days later she became anorexic and the next day developed deep rapid respirations. On the day of admission she was extremely lethargic, requiring assistance on her many trips to the bathroom because of a staggering gait. The eyes became "crossed," and there was drooping of the left upper eyelid. In the early afternoon there was a brief episode of left-sided epistaxis.

Physical examination revealed a thin white child in borderline contact, appearing acutely ill, with deep rapid breathing and flaring of the nasal alae. The temperature was 96 F, the pulse 120, respirations 38. The blood pressure was 85/50. The skin was dry, cold, and pallid, showing poor turgor but no cyanosis except in the lips. The left eye was ptosed, and the globe pointed straight

ahead with a dilated, fixed pupil. There was facial weakness and an absent corneal reflex on the left. Both fundi were normal. The left nostril was filled with a bloody mucoid discharge. Fine moist rales were present at the base of both lungs posteriorly. Reflexes were absent except for weakly positive ankle jerks bilaterally. Kernig's and Brudzinski's signs were absent. There was paralysis of the left oculomotor, trochlear, abducens, trigeminal, facial, and hypoglossal cranial nerves.

Urinalysis showed a pH of 5.0, sugar 4+, and acetone 4+. The hematocrit was 32%; WBC 40,000, with 75% segmented neutrophils. The blood sugar was 460 mg. %, the carbon dioxide 5.2 mEq. per liter, and the sodium 128 mEq. per liter.

On admission the patient was given regular insulin 20 units intramuscularly and 20 units subcutaneously, plus 2,000,000 units of aqueous penicillin intravenously. Fluid and electrolyte infusions were started. Six hours later the temperature rose to 100 F. The urine continued to show 4+ sugar and 4+ acetone. Nine hours after admission a noticeable increase in the left periorbital swelling was observed. Fifteen hours after admission the patient was completely out of contact. Shortly thereafter respirations ceased, and the heart beats became feeble. Following a favorable response to artificial respiration and oxygen, the child vomited 250 cc. of reddish-brown guaiac-positive fluid. A lumbar puncture performed at that time revealed a ground-glass-appearing fluid containing 1000 cells per cubic millimeter, with 95% neutrophils, 5% monocytes, protein 58, sugar, 268 mg. %, and chlorides, 116 mEq. per liter. The blood sugar at that time was 326 mg. %. A few hours later the Babinski and Chaddock signs were present, and a definite bluish hue to the entire left face was noted. Chloramphenicol (Chloromycetin) 1 gm. IM every 12 hours was added to the therapeutic program.

X-rays of the sinuses at this time revealed thickening of the membranes lining the left maxillary, ethmoid, and sphenoid sinuses, without evidence of fluid levels.

Eighteen hours after admission it was noted that the left retinal artery was thrombosed and that the child had developed a right hemiparesis. Six hours later the left frontal, ethmoid, and sphenoid sinuses were explored under local anethesia. Bloody fluid was found in the ethmoid and sphenoid sinuses, and the membranes were swollen and hemorrhagic. Of particular interest at operation was the dry, bloodless, black appearance of the left inferior and middle turbinates. During the first 24 hours of hospitalization, fractional urines continued to show 4+ sugar and 4+ acetone in spite of heavy doses of insulin. Quite suddenly both sugar and acetone disappeared from the urine, and the patient required no insulin during the following 29 hours of life. At the same time the blood sugar fell to within normal limits. Erythromycin, 250 mg. IM every six hours, was added to the therapeutic program. Despite all efforts, including norepinephrine and plasmin, the patient steadily declined and died 53 hours after admission.

At autopsy, the left middle and inferior turbinates were dry and black (Fig. 4), with necrosis of the epithelium on microscopic examination. The subepithelial tissues were infiltrated by vegetative mycelia of mucormycosis (Fig. 5). In the spaces between the turbinates the aerial or reproductive mycelia were seen (Fig. 7). This fungus was characterized by broad nonseptate hyphae. The ovoid sporangia were filled with sporangiospores.

The left internal carotid artery in the cavernous sinus area was distended by a thrombus, which was gray in some areas and deep red in others. Microscopic examination of the artery revealed necrosis and mycelial invasion of its wall, and an antemortem, mycelia-filled thrombus in its lumen (Fig. 6).

Microscopic sections of the eye revealed acute necrosis of the retina. The retro-orbital tissues showed multiple thrombi and fat necrosis, with mycelia invading the nerves and the extraocular muscles.

The left cerebral hemisphere was larger than the right. The inferior surfaces of the left frontal and temporal lobes were soft and necrotic, and there was localized thickening of the dura. The left side of the pons was soft, and the overlying meninges were cloudy. Cultures of the necrotic areas produced no bacterial growth. Microscopic sections showed areas of necrosis in the pons, cerebellum, frontal and temporal lobes. Acute inflammation was present in the involved meninges. All sections of these tissues showed thrombi of small vessels, with mycelia throughout the brain tissue and blood vessels.

Case 3.—A 57-year-old diabetic white woman was brought to the hospital in an unresponsive state.

The present illness began 48 hours prior to admission with the onset of anorexia and nausea. Twenty-four hours later she was observed to be confused and disoriented. There was no history of trauma.

The patient was a known diabetic for 25 years, well regulated with 40 units of isophane insulin daily. The right great toe had been amputated three years prior to admission, with intermittent episodes of drainage since that time, the last ocurring three weeks prior to admission.

Physical examination on admission revealed a pale white woman appearing chronically ill and responsive only to painful stimuli. The blood pressure was 110/70, the pulse 100, and the temperature 99 F. The respirations were 24 and not Kussmaul in character. The lips were dry and

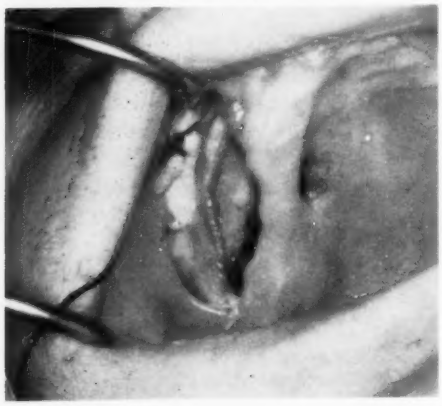
crusted; the tongue was dry, and the odor of acetone was present on the breath. The right pupil was larger than the left, and lenticular opacities precluded examination of the fundi. The lungs were clear to percussion and auscultation. The right great toe was surgically absent, with purulent discharge from the stump.

Urinalysis showed 1+ albumin, 4+ sugar, and 4+ acetone. The white blood count was 18,000, with 88% neutrophils. The blood sugar was 960 mg. % and the CO_2 13.0 vol. %. The spinal fluid examination was normal.

On admission the patient was given procaine penicillin and streptomycin. Insulin and electrolytes were started for correction of the acidosis. Twenty-four hours later it was noted that the patient's mental status had not improved as expected with regulation of the acidotic state. Neurological examination revealed moderate edema of both sclerae. The extraocular muscles were grossly normal in all directions, with slight limitation in lateral movements of the right eye.

On the fourth hospital day, right infraorbital and periorbital edema with tenderness was observed. Twenty-four hours later an eroded area was seen on the right maxillary alveolar ridge and hard palate communicating with the maxillary sinus (Fig. 8). Cultures of the eroded area grew out streptococci and nonhemolytic Staphylococcus aureus. At that time the facial swelling became erythematous and indurated, and the excursions of the right eye reduced in all directions. Aqueous penicillin was added to the antibiotic program. X-rays of the sinuses revealed bilateral maxillary sinus cloudiness without evidence of fluid level. A chest x-ray revealed linear atelectasis and compression of the right lower lobe with minimal infiltration of the right base. Scrapings of the oral ulcerations were negative for fungi.

Fig. 8 (Case 3).—Photograph showing extension of the disease into oral cavity.



On the seventh hospital day the right maxillary sinus was aspirated and irrigated. Bloody crusts were observed in both nostrils. Very dark bloody fluid was obtained from the antrum. There was no evidence of pus in the nose, sinuses, or oral lesions. Cultures of the fluid grew out α-Streptococcus and Aerobacter aerogenes. Mycology studies were again negative. The aqueous penicillin was discontinued, and the procaine penicillin increased to over 6,000,000 units per day.

On the 12th hospital day penicillin and streptomycin were discontinued and nystatin (Mycostatin) started. Twenty-four hours later the right seventh cranial nerve showed weakness. The diabetic state, which had been easy to control since initial regulation, suddenly became difficult to manage. At this time the right maxillary sinus was stripped of its lining, and wet preparations for fungus showed the presence of what appeared to be Rhizopus. The fungus containing tissue was repeatedly cultured, but no growth on laboratory media was obtained. Histologic preparations from the sinus lining showed evidence of active tissue invasion by the large nonseptate hyphae (Fig. 9). With the diagnosis of mucormycosis established on a histologic basis, all antibiotics were discontinued and the patient started on intravenous and topical amphoteracin. A chest x-ray at this time revealed an abscess in the right lower lobe.

On the 20th hospital day the patient complained of severe headache and of photophobia and decreasing vision in the right eye. At this time a large ulcer was observed in the left hard palate mucosa. The next day bronchoscopy was performed, and wet preparations of the secretions revealed the presence of broad nonseptate hyphae. Repeated cultures of this material failed to grow out the fungus.

After the 34th hospital day the patient started on a progressive downhill course with increased confusion and lethargy, and died on the 42d hospital day.

A routine autopsy was performed. No tissue was obtained from the nose, sinuses, oral cavity, or orbit. The areas were carefully cultured for fungi, but no growth was obtained. Gross examination of the cavernous sinuses, internal carotid arteries, meninges, and brain was unremarkable. Multiple abscesses were seen in the lower and middle lobes of the right lung. A granular yellow-green thrombus was present in the right middle lobe artery, occluding its entire lumen. An area of infarction was also present in the right middle lobe. Abundant hyphae and spores were seen in sections of the abscess cavity walls, the area of infarction, and the artery thrombus. The most luxuriant area of growth was observed in sections made of the thrombus. Sections of the other body organs including the pituitary, failed to show evidence of invasion by the hyphae or spores.

Fig. 9 (Case 3).—Section of maxillary sinus lining showing active invasion by hyphae.

Comment

Our first case of mucormycosis in 1954 was considered an interesting example of tissue necrosis related to the necrotizing toxin of Staphylococcus aureus complicating a case of diabetes. At autopsy, routine tissue sections from the area of infarction and the thrombosed internal carotid artery failed to identify the causative organism. The peculiar dark dried blood at the anterior end of the turbinates proved to be an area of infarction.

Our second case also had what looked like a bloody nasal discharge, but it could not be removed from the turbinate by careful cleansing with peroxide. Thorough examination of the nasal area at surgery revealed a complete infarction of the inferior turbinate. Sections of this turbinate and of the internal carotid artery obtained at autopsy provided the diagnosis. Thus our second case of this peculiar necrosis of the turbinate in a diabetic patient led to a review of the tissues obtained from our first case, and discovery of hyphae and spores in both the turbinates and internal carotid artery.

The diagnosis of mucormycosis in our third case was made prior to death but not until the disease had extended from the nose and sinuses into the oral cavity. The disease was suspected shortly after admission, but several attempts to culture the fungus from the nose and oral cavity failed to confirm our clinical impression. Scrapings from the oral lesions were repeatedly examined without either hyphae or spores being found. A diagnosis was finally obtained by histologic examination of the maxillary sinus mucosa. At operation the whole lateral nasal wall was absent, indicating that the disease had existed in the turbinates long before the oral cavity and sinuses were invaded.

The onset of cerebral mucormycosis during the course of a serious preexisting illness may be overlooked. The following signs and symptoms frequently usher in the disease and are probably pathognomonic when developing in a patient whose diabetes is out of control, or has been recently:

1. Blood-tinged nasal discharge on the side of facial pain, of brief duration, and usually very dark in color
2. Soft periorbital or perinasal swelling which progresses to induration and discoloration (with progressive vascular occlusion)
3. Ptosis of the lid and proptosis of the globe, dilatation and fixation of the pupil, limitation of global mobility

4. Lethargy, progressive, in spite of good chemical response to diabetic therapy
5. Black necrotic turbinates, easly mistaken for dried blood
6. Loss of corneal reflex and onset of facial weakness, usually seen late in the course of invasion

Other findings, such as unilateral headache, sinus pain, fever, and patchy clouding of sinuses on x-ray, usually accompany a developing mucormycosis but are less specific than those listed above. The findings listed may not all be present in any given case or occur in the order described. Any of them, however, in an uncontrolled diabetic, calls for a thorough ENT examination and a tentative diagnosis of mucormycosis until proved otherwise.

The most striking physical finding to the otolaryngologist is the black discoloration of the nasal mucosa, unlike anything seen in other nasal diseases and resembling a dried mass of clotted blood. This color as previously stated is apparently due to infarction of the turbinate as a result of extensive invasion and occlusion of the regional arteries by the hyphae. The histologic demonstration of broad-branching nonseptate hyphae, actively invading the submucosal layers and showing particular affinity for arteries, readily differentiates this lesion from idiopathic midline granuloma, thrombosed mucous polyps, rhinoscleroma, tuberculosis, syphilis, rhinosporidiosis, benign and malignant tumors.

Once the clinical signs and symptoms suggest mucormycosis, it is essential that tissue evidence of the disease be obtained. Cultures should be taken repeatedly for fungi, but early disappearance of the fungus from the mucosal surface often makes this an unrewarding task. Biopsy should be performed on necrotic areas in the nose. Sinus x-rays should be examined carefully for localized or diffuse edema of the mucosal lining and for evidence of bone destruction. Biopsy should be done on any areas suggestive of invasion. Should a suspected case of mucormycosis come to autopsy without a tissue diagnosis or a positive culture, special consideration must be given the nose, sinuses, oral cavity, and orbits because tissues are not usually obtained from these areas for routine histologic examination.

Harris[2] could credit no specific therapy for the amazing recovery of his case. He felt that precise control of the diabetes, use of iodides, desensitization, and the avoidance of prolonged antibiotic therapy were favorable factors. The principles of therapy are well known, particularly in blastomycosis, where Smith[23,31] has shown the adverse effect of a hypersensitive state on the course of the disease, especially during therapy with iodides. We must stress the fact that although ocular signs are usually reported to be the earliest clinical manifestation of the disease, nasal and sinus abnormalities occur earlier and must be searched for as soon as the diagnosis is suspected.

Summary

Cerebral mucormycosis, a rapidly fatal fungus disease, frequently begins in the nose, spreads quickly to the sinuses or orbits on the way to the brain, and produces striking but characteristic physical changes around the nose, eyes, and mouth. The causative organism, Rhizopus, is nonpathogenic ordinarily, but becomes invasive in the presence of certain illnesses being treated with chemotherapeutic agents. The antileukemic preparations, the corticosteroids, and particularly the antibiotics used in treating bacterial infections in diabetics are notable predisposing agents in the development of cerebral mucormycosis.

Early recognition of the disease during its nasal, sinus, or orbital stages has led to cure in several previously reported cases.

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Tracheostomy*

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Introduction

Airway obstruction is one of the few real emergencies in medical practice. This illustrated article demonstrates the anatomy related to and the operative technique of tracheostomy. The indications for a tracheostomy are divided into clinical and anatomic. The clinical indications are further seperated into absolute and select groups while the anatomic is separated into supraglottic, glottic, and subglottic locations of airway obstruction. The resultant advantageous changes in respiratory physiology following tracheostomy are enumerated and cannot be overemphasized. Postoperative care is most important and is listed in detail.

The indications and technique of tracheomediastinotomy (combined tracheostomy and bilateral cervical mediastinotomy) are described.

It is urged that a policy of watch and wait, except in rare instances as in the newborn, should give way to the acceptance of tracheostomy not as a last-minute resort but as a well-executed but yet simply performed procedure.

Indications, Clinical

I. Absolute
- A. Radical Head and Neck Surgery
  1. Commando
  2. Bilateral radical neck dissection (1 or 2 stages)
  3. Extensive pharyngeal or oral surgery
- B. Laryngeal Surgery
  1. Cordectomy
- C. Thyroid Surgery
  1. Total thyroidectomy with sacrifice of both recurrent nerves
  2. Preexisting abductor cord paralysis—unilateral or bilateral
- D. Severe Injuries
  1. Larynx
  2. Pharyngoesophagus
  3. Trachea
  4. Stove-in chest
  5. Oral cavity
  6. Facial bones
- E. Severe Burns of Air Passages
- F. Severe Barbiturate Poisoning
- G. Progressive Laryngeal Edema
  1. Inflammatory—e. g., laryngotracheitis
  2. Trauma
  3. Pressure—e. g., postoperative hematoma
- H. Bilateral Abductor Cord Paralysis
- I. Radiation Therapy
  1. Bulky tumors of larynx or pharynx
  2. Preexisting abductor cord paralysis—unilateral or bilateral
- J. Severe Intracranial Injuries and Vascular Accidents
  1. Unconscious over 24 to 48 hours
  2. Absent cough reflex
  3. Collapse of airway
- K. Infections
  1. Bulbar poliomyelitis
  2. Diphtheria
- L. Tumefactions Comprising Airway

II. Select Use
- A. Mediastinal Emphysema
- B. Micrognathia
- C. Cardiac Arrest

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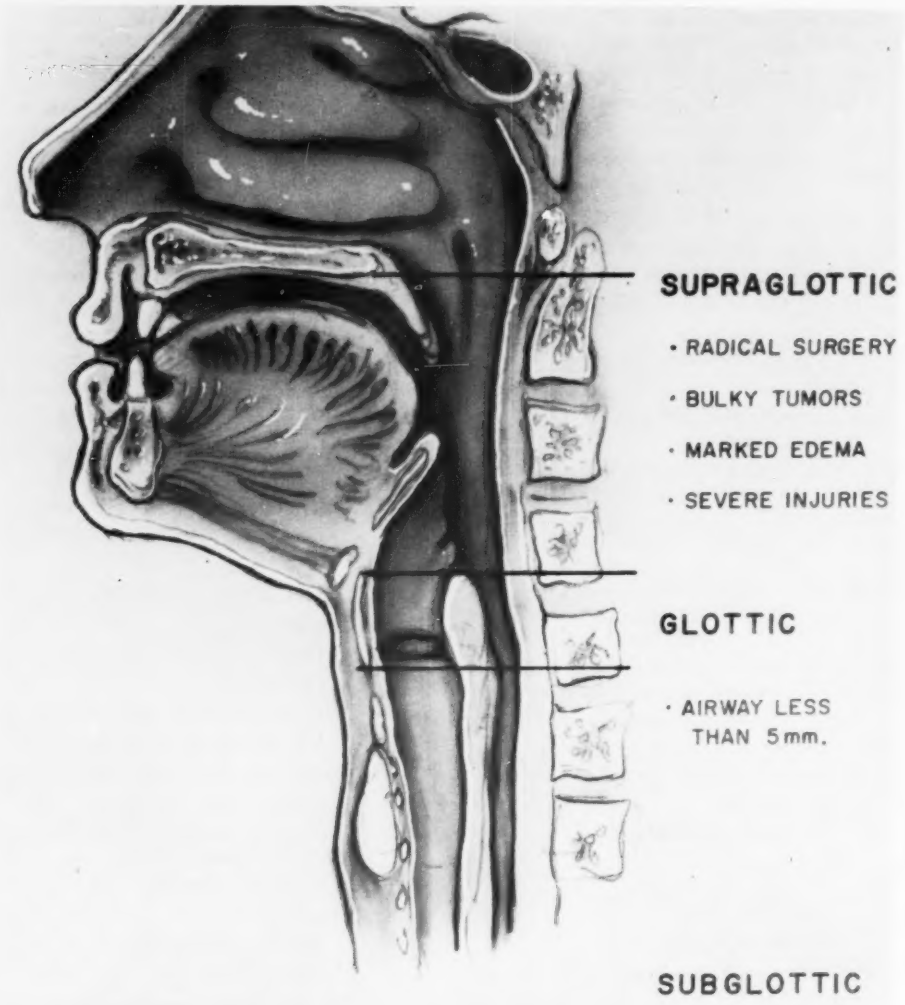
D. Difficult Tracheal Toilet (from whatever cause)
E. Myasthenia Gravis
F. Reasonable Doubt (except in infants)
G. Infections
   1. Tetanus
   2. Rabies
   3. Angioneurotic edema
H. Thoracic Surgery
I. Inhalation of Caustics

Indications, Anatomic
Diagnostic Importance of Anatomic Location

1. Determines site of election; low tracheostomy through sternum may be required
2. Air hunger may not be relieved by tracheostomy, e. g., vascular ring compressing trachea



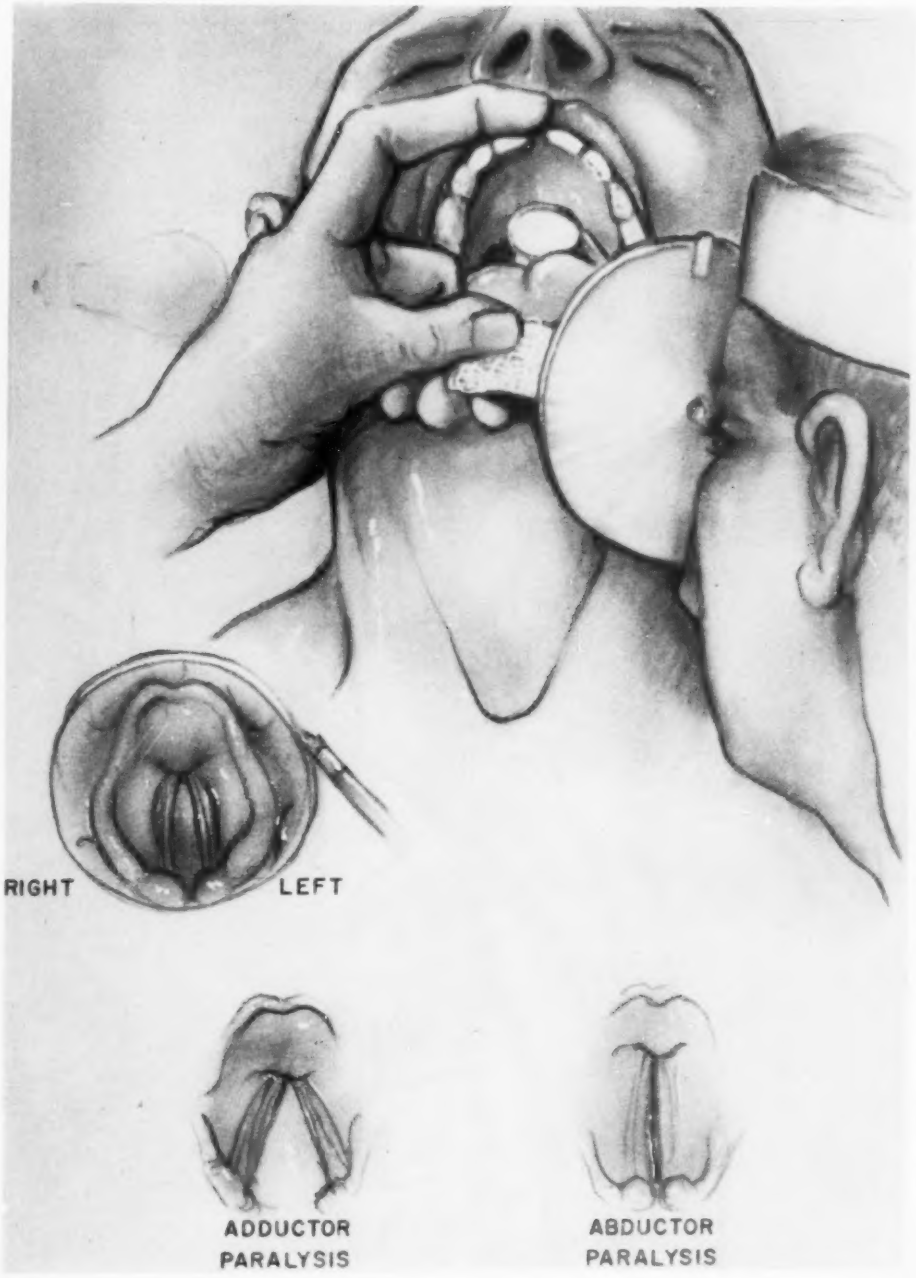
- EXCESSIVE AND INSPISSATED TRACHEOBRONCHIAL SECRETIONS
- EXTENSIVE CHEST INJURIES AND SURGERY
- TRACHEAL TUMEFACTIONS ABOVE SITE OF ELECTION

Diagnosis of Anatomic Location

1. Indirect laryngoscopy
2. Direct laryngoscopy
3. Bronchoscopy
4. Tracheogram and bronchogram

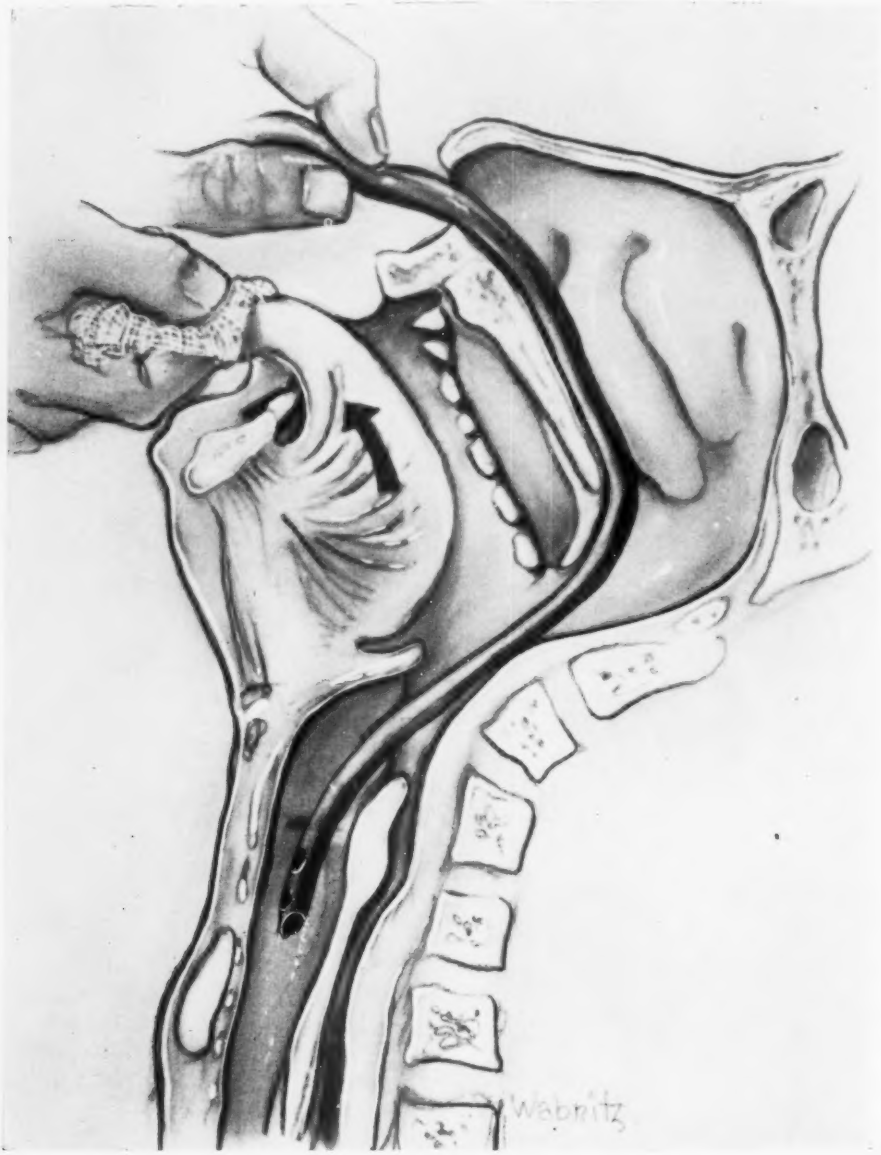
Instruments for Indirect Laryngoscopy

1. Head mirror and lamp or head light
2. Laryngeal mirror
3. Hot water or alcohol lamp
4. Gauze



Emergency Establishment of Airway

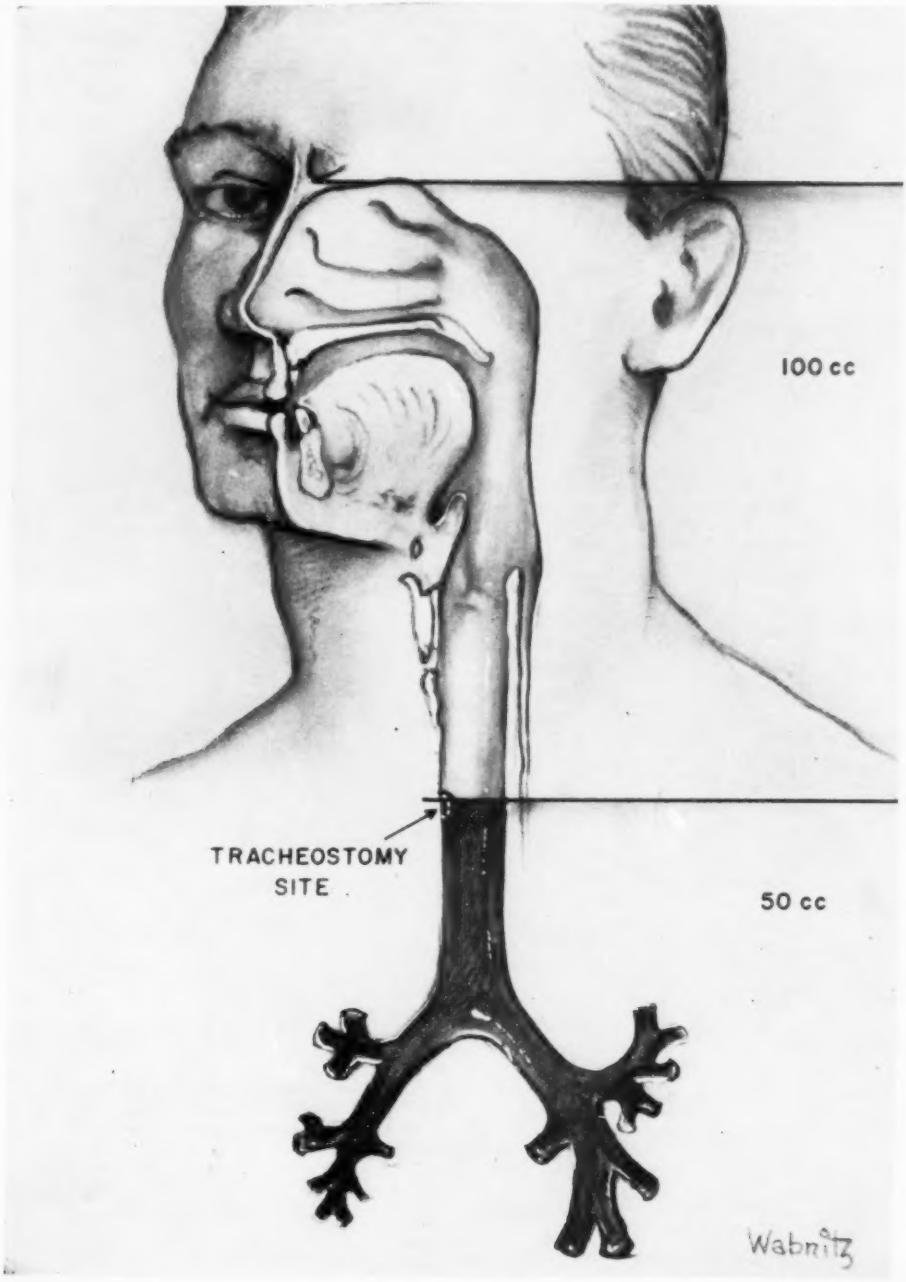
1. Direct laryngoscopy and insertion of bronchoscope or endotracheal tube
   (*a*) Very rapid
   (*b*) Allows leisurely tracheostomy while patient is receiving oxygen
2. Insertion of bronchoscope directly
3. Insertion of nasotracheal tube—any tubing can be used—e. g., stethoscope
4. Incision in cricothyroid membrane should be avoided
5. Percutaneous insertion of large-bore needle into trachea and administration of oxygen—only if other instruments not available
6. Various mechanical devices for tracheostomy—only if other instruments not available



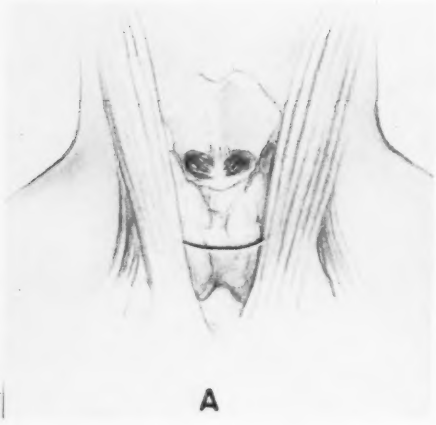
Physiology

Changes Following Tracheostomy

1. Lessens dead space by 60% (total dead space 150 cc.)
2. Ventilation improved
3. Aids stabilization to chest wall
4. Reduces hypercapnia
5. Aids correction respiratory acidosis

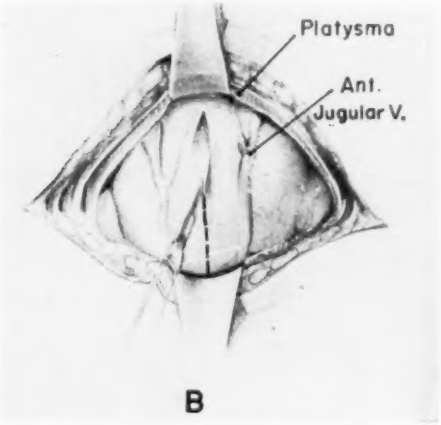


Technique for Tracheostomy



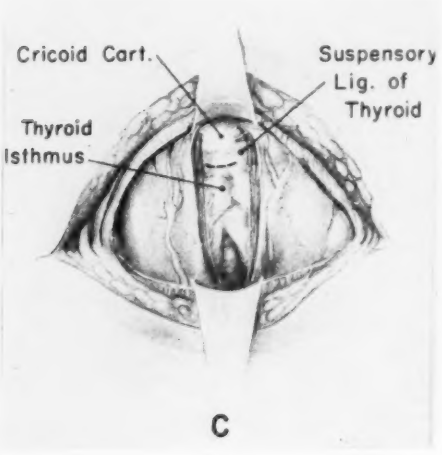
A

A, with the neck hyperextended, the cricoid cartilage is palpated and then a 4 cm. to 6 cm. horizontal incision below the cricoid and about two fingers' breadth above the sternoclavicular junction is made along a skin crease.



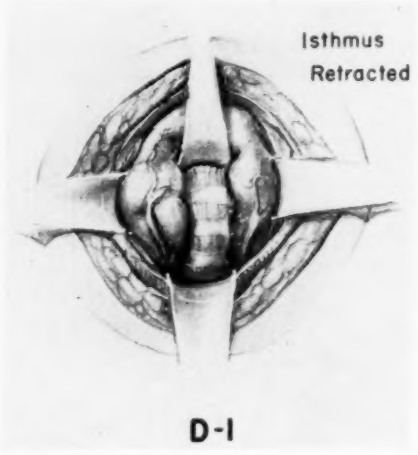
B

B, this horizontal incision is carried through the superficial fascia and platysma muscle. The flaps thus formed are separated above and below from the underlying fascia. A vertical midline incision is then made through the fascia which envelops the strap muscles.



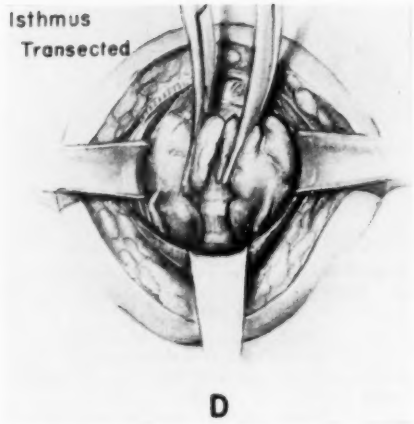
C

C, the cricoid cartilage is identified. Just below it, the suspensory ligament of the thyroid gland and thyroid isthmus are noted.

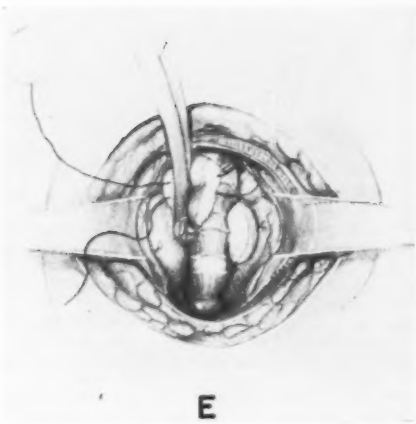


D-I

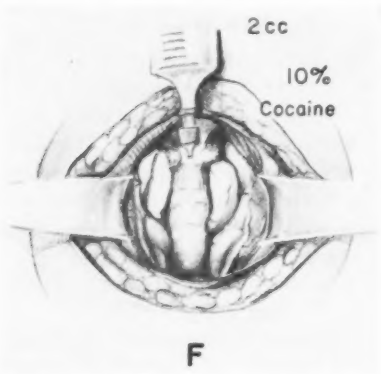
D-1, the strap muscles are retracted laterally and the thyroid isthmus retracted upward, if possible, exposing the trachea in the region of the third tracheal ring.



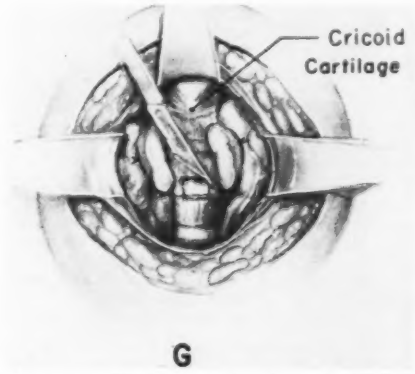
D, if the isthmus cannot adequately be retracted, a small transverse incision is made through the suspensory ligament (Fig. *C*). Medium-sized curved clamps are inserted through this opening in the suspensory ligament separating the isthmus from the trachea. The isthmus is doubly clamped and transected.



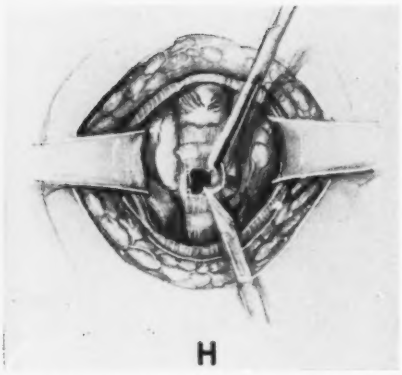
E, the transected ends of the isthmus are suture ligated, preferably with 2-0 black silk.



F, a hypodermic needle is inserted into the trachea; air is withdrawn to be certain that the needle is in the tracheal lumen, and 2 cc. or less of 10% cocaine instilled into the tracheal lumen. This reduces the troublesome coughing when the tube is inserted.



G, the second, third, or fourth tracheal ring is identified and using a No. 11 blade knife a 5 mm- to 10 mm. horizontal incision is made directly above the selected cartilaginous ring.



H

H, the incision is carried downward across the ring and then back horizontally directly below the ring. An Allis clamp is gently affixed to the cut end of the cartilaginous ring, which is then transected. A small portion of the ring is thus removed. (If preferred, a small skin hook may be used in lieu of the Allis clamp; however, this may tear and fragment a section of cartilage with the danger of aspiration.) The airway is now established, and, if necessary, tracheal toilet begun even before insertion of the tracheostomy tube. If an emergency establishment of airway was previously performed using an endotracheal tube or bronchoscope (which is often used in children), the tube or bronchoscope is now removed.

I

I, the previously made ready tracheostomy tube (No. 6 for adults) is now inserted into the trachea and the obturator quickly removed. The end of the tube should never reach the carina.

J

J, the skin edges are lightly approximated with Dermalon. A small space should be left at either side of the tube to minimize the danger of tension emphysema. The tube is both sewn to the skin and affixed with tape tied with a square knot behind the neck. The inner tube is then inserted. A moist piece of plain gauze is then hung over the tube opening.

Remove Small Section Tracheal Ring

1. Minimizes danger of cartilage foreign body
2. Minimizes infolding of tracheal wall
3. Immediately establishes airway without forcing tracheostomy tube into trachea
4. Helps prevent tracheal granulomas which are so serious in children
5. If tube becomes dislodged or is removed, airway can be reestablished by simply retracting wound edges, and reinsertion is easier

Postoperative Care

Basic Principles

I. Keep moist
II. Deep suction
III. Keep clean

1. Keep flap dressing moist
2. Aerosol oxygen or cold steam with or without detergents 20 minutes every one to two hours or continually as required
3. Remove and cleanse inner tube every half to one hour
4. Deep rapid suction every hour and as necessary
5. Drops of sterile normal saline or water every hour and as necessary when secretions become thick
6. Change flap dressing as necessary
7. Avoid frayed edges on dressing
8. Avoid leaving suction catheter in tracheostomy tube beyond a few seconds
9. Use separate suction catheter for oral cavity as necessary

10. Early instructions to patients for self care

Postoperative Advantages

1. Ease of tracheobronchial toilet
2. Ease of oxygen administration
3. Ease of instillation of antibiotic directly into trachea
4. Conscientious but not skilled medical personnel necessary

Warning and Recommendations

A. High Morbidity and Mortality in Infants under 9 Months of Age
   1. Difficult to maintain tube patent
   2. Tendency to obstruction granulomas of trachea
   3. Almost impossible to extubate until over one year of age

B. Not a Panacea for All Airway Problems

C. Perform as Elective Rather than Emergency Procedure

D. Be Certain Tubular Structure Selected is Trachea and Not Large Vessel

E. Be Aware of Tracheal Casts or Plugs Distal to Tracheostomy Tube—Remove Tube Immediately to Extricate Cast or Plug

F. Suture Skin Loosely Around Tube—If Tight May Cause Mediastinal Emphysema

G. Tube Must Not Reach Carina

H. Sew Tube to Skin

I. Use Deep Suction Beyond End of Tube—Remove Rapidly

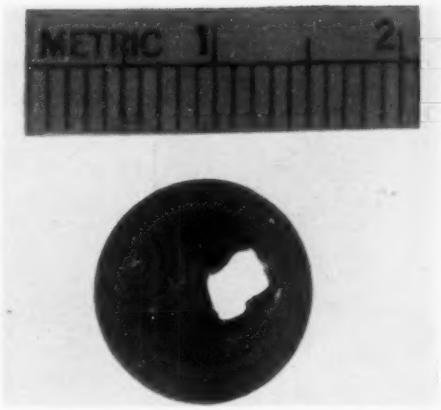
J. Follow Emergency Establishment of Airway with Operative Tracheostomy

Tracheomediastinotomy

Traumatic injuries to the larynx, pharynx, trachea, esophagus, and chest may cause mediastinal emphysema. If tension develops, compression and occlusion of the venous return to the heart may occur. Unilateral or bilateral pneumothorax may also occur with rupture of the mediastinal pleura.

Treatment consists of

1. Tracheostomy
2. Bilateral cervical mediastinotomy
   = Tracheomediastinotomy

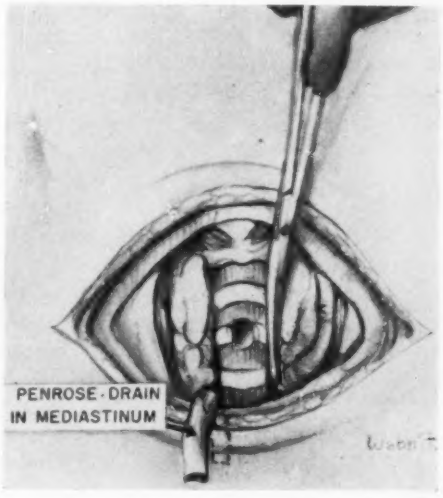


Compromised endotracheal tube left in situ 24 hours despite excellent nursing care.

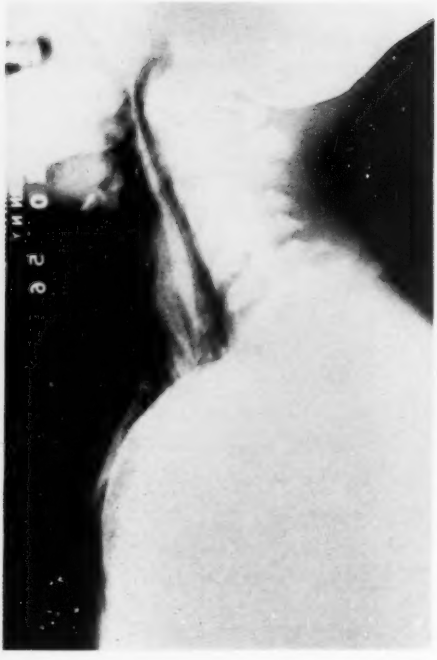
3. Intercostal underwater drainage if pneumothorax present

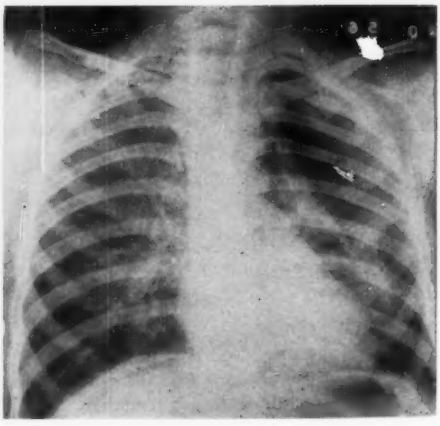
Technique of Tracheomediastinotomy

1. Usual tracheostomy
2. The mediastinum is entered by carefully and gently inserting a large curved hemostat alongside each side of the trachea down to the esophagus for several centimeters below the level of the clavicles. The instrument is opened slightly and Penrose drains inserted bilaterally. The tracheostomy tube is inserted and skin edges are only partially approximated.



Mediastinal emphysema associated with unilateral vocal cord paralysis following trauma to the larynx.
Treatment: tracheomediastinotomy.





Mediastinal emphysema, subcutaneous emphysema, and pneumothorax following trauma to the neck in an automobile accident.
Treatment: Tracheomediastinotomy and intercostal drainage.

Secretory Otitis Media in Children

PIERCE W. THEOBALD, M.D., Chicago

The history of secretory otitis media dates almost as far back as the history of otology as a specialty; yet secretory otitis media is perhaps not the correct term for this disease entity. Fluid in the middle ear and adnexa may be either a transudate or an exudate. Although Eggston and Wolff [1] have noted that goblet cells may appear in the middle ear mucosa in the presence of infection, there is little evidence that middle-ear fluid is secreted in the strict sense that word implies, at least in the early stages of the disease. Among the several other terms used to describe the condition is serous otitis media. However, the fluid may be either serous or mucous, or any and all gradations between the two. Through common usage, as with so many other disease processes in the science of medicine, secretory otitis media seems to have become the most generally accepted term.

Both the real and the apparent increase in the incidence of the disease will be pointed out later. As has so often been repeated, the condition was first described by Politzer [2] in 1869 and thoroughly narrated in his textbooks. However, except for the descriptions of Kyle,[3] Bezold,[4] Ballenger,[5] Phillips,[6] and Kerrison,[7] in their respective texts, the subject received little or no attention in the literature, except by Lawson,[8] until 1940. As Watkins [9] pointed out, the initial period of early interest was followed by an almost total lapse of study of secretory otitis media until the onset of World War II. At that time the consequences of high-altitude flight and rapid changes in altitude and barometric pressure due to increased performance of aircraft resulted in a marked increase in the incidence of aerotitis, a distinct clinical entity described and named only as recently as 1937 by Armstrong and Heim.[10] The problem of aerotitis, the purest form of secretory otitis, stimulated investigation in the field of fluid in the middle ear not related to flying.

Jones,[11] Robison,[12] Hoople and Blaisdell,[13] and Hantman [14] were among the forerunners of those who restimulated interest in the subject of fluid ears. In fact, the pendulum has swung the other way. Ivstam,[15] writing from Sweden, stated that "secretory catarrh is believed to be such a serious problem (in the United States) that Hoople termed it a 'challenge to otolaryngology'." That the problem is a serious one, not so much from the standpoint of the disease itself as in the light of adequate and accurate diagnosis and treatment, can hardly be denied. Hoople,[16] in a truly monumental work in 1950, drew attention to the lack of teaching concerning secretory otitis media in our medical schools, in our modern textbooks, and in postgraduate courses. To him, and to Suehs [17] for his exhaustive study in 1952, a great deal of credit must be given for the awareness of the possibility of its presence and the greater vigilance of otologists in recognizing and diagnosing it. Part of the battle has been won because of their efforts and foresight, but much still needs to be done in order to conserve the hearing of an alarmingly large segment of the younger population.

Several years ago I wrote concerning my experience with secretory otitis media and drew several conclusions. Since that time the increase in number of cases I noted has continued, but more recently there seems to have been an increased incidence of the

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condition in children, which seems definitely related to enlarged and/or infected adenoid tissue, and to frequent and often indiscriminate use of antibiotics. The literature pertaining to secretory otitis in children is rather sparse; yet the importance of adequate hearing in young people cannot be overstressed. No one can deny that hearing defects are especially serious in children. As Schenck [18] has pointed out, hearing loss of youngsters may go undetected for a long period, particularly at a time when adequate hearing is so necessary and important to normal progress and learning. The increased incidence of the disease in the younger age groups has stimulated me to a greater interest in the problem. The purpose of this paper is to review the literature on secretory otitis media, especially as it pertains to children; to record and evaluate my own experience with the disease; and to comment and attempt to draw conclusions concerning methods for case-finding, diagnosis, treatment, and, it is hoped, prevention of the disease itself or at least prevention of the subsequent irreversible hearing loss it can, and many times does, cause.

That there exists a real, as well as an apparent, increase in the incidence of secretory otitis media, I believe to be a fact, and that belief is shared by others. Hall [19] and Tremble [20] feel there is an increase in frequency of it, and place the blame squarely on the increased use of antibiotics. Schenk [18] shares their view in that he feels the prevention of hearing loss following acute otitis media has become more complex since the introduction of antibiotics. Jones [21] reports he is finding an increasing number of cases. Eagle [22] implicates virus infection and notes increasing incidence. Hall [19] and Lederer [23] refer to hypovirulent infections. Suehs,[17] Jordan,[24] and Hoople [16] feel that increased awareness of the disease and vigilance of otologists in diagnosing it accounts for the increased incidence. However, Hoople [16] also stated that he has noted a 20% increase in incidence since the advent of chemotherapeutic and antibiotic drugs, and, more important, he found most of the increase in children. Robison and Nicholas,[25] Samuels,[26] Rentschler and Gibbs,[27] and Tremble [20] have also noted increased incidence to a greater degree in children. Woodward [28] feels the increase is due to the two principal factors of vigilance of otologists and increased use of antibiotics, a view shared by the author.

That the primary factor in the etiology of fluid in the middle ear is Eustachian tube blockage there is little doubt. However, as Hoople [16] and others have pointed out, the blocked tube in itself is not the complete answer, for not every blocked tube is accompanied by fluid in the middle ear. Jordan,[24] Derlacki,[29] and others have implicated allergy rather strongly. Rentschler and Gibbs [27] stated that, since the middle ear mucosa is continuous with that of the nose and pharynx, it is reasonable to assume that its response to certain allergens would be similar. Hilger [30] and Williams [31] have pointed out the relationship of autonomic dysfunctions to vascular changes and allergy. Boswell [32] discusses the evidence pointing to a typical vascular reaction as the inevitable autonomic response to environmental stress in our modern fast-paced world, and feels that the emotional stress caused by unusual fear and anxiety raises the sensitivity level of our psychosomatic mechanism and results in allergic manifestations. Suehs [17] pointed out the relationship between incidence of the disease and altitude and humidity, there being a greater number of cases reported from areas of low altitude and high humidity. He also stated that the disease was more prevalent in young children because they are predisposed to tubal obstruction and more prone to upper respiratory infection.

In the series of children being reported Eustachian tube blockage was by far the most important single factor to be reckoned with. Bezold [4] referred in his textbook to the gathering of serum in the spaces of the middle ear as a direct consequence of occlusion of the tubes which hardly ever is lacking after prolonged occlusion. Fowler [33] feels that tubal occlusion is responsible for

28% of deafness in children, as contrasted with 80% of adult hearing loss. I feel that 28% is a low figure, at least in my experience with the causes of hearing loss in children. Singleton [34] has said that in general any condition that contributes to swelling and closure of the Eustachian tube may be a factor in the production of serous otitis media. He went on to name the many and varied conditions, such as acute and chronic upper respiratory tract infections, allergy, dental malocclusion, and numerous other factors, occurring less frequently, that can cause tubal swelling. He mentioned hypertrophied and diseased adenoids as a frequent offender in children. He also implicated early chemotherapeutic treatment of otitis media resulting in sterilization of the middle-ear infection as a cause. These last two factors are considered of primary importance in the etiology of secretory otitis media in children, so far as my experience is concerned, and their indictment will be elaborated upon later in the discussion. Woodward [28] goes so far as to say that the most frequent cause of conductive deafness in children is the result of antibiotic-treated otitis media. Many authorities will agree with him.

Regardless of the underlying and basic etiology, most authors readily agree that the inciting cause of secretory otitis is blockage of the Eustachian tube, or failure of the tube to open properly to ventilate the middle ear. Rentschler and Gibbs [27] feel that the epithelium lining the middle ear is similar to the lung alveoli in its ability to absorb oxygen, a suggestion originally made by Bezold.[4] Through failure of the Eustachian tube to open intermittently to allow replacement of the absorbed oxygen, a negative pressure develops and persists in the middle ear. Schechter,[35] in discussing aerotitis, drew attention to the delayed occurrence of it in aviators who have been breathing pure oxygen over long descents. Matsumura [36] studied the composition of air in the tympanic cavity and found a correlation between the oxygen percentage and the patency of the Eustachian tube. He found the oxygen value lower when the tube was obstructed. Correlation between oxygen percentage and negative pressure in the tympanic cavity was suggested, but not definitely proved. If the above theories are accepted as fact, then it can be assumed that the next step in the development of secretory otitis is the accumulation of fluid in the middle ear. From this point on a vicious cycle develops, for I feel that fluid in the middle ear causes edema of the tube to persist. The cycle can rarely be broken except by myringotomy.

The nature and content of the fluid in the tympanum has been extensively discussed by many authors. Disagreement still exists as to whether it is an exudate or a transudate, or a combination of the two, and as to the protein content. Bezold [4] considered the fluid to be a transudate. He noted that it may become more viscid if it stays in the middle ear for some time. Ivstam [15] states it is possible to distinguish between transudate and exudate by chemical analysis. However, Cantarow and Trumper [37] state the protein content of transudates and exudates varies in different parts of the body and depends on variations in the inflammatory process and capillary permeability, as well as water absorption. Suehs,[17] Tremble,[20] Hoople,[16] Robison,[12] and others have honest differences of opinion as to the formation and content of the fluid, perhaps partially due to the result of differences in methods of collection and analysis. Further, the controversy is reflected by various authors in the literature throughout the history of this disease from the time of Politzer. The presence or absence of eosinophils is the subject of another great controversy. Koch,[38] Derlacki and Shambaugh,[39] Jordan,[24] and Ivstam [15] report finding eosinophils in the fluid and attach importance to the finding. Robison and Nicholas,[25] and Suehs [17] found none. Ivstam [15] found eosinophilia in the fluid from cases of chronic secretory otitis, but was unable to produce positive intracutaneous tests for allergy or demonstrate findings of allergic rhinitis. He nevertheless

felt that cases demonstrating eosinophilia were allergic in origin. Hansel [40] feels that eosinophils are a most dependable sign of allergy, but, on the other hand, Petersen [41] has presented evidence that eosinophils may be produced at will as a result of localized tissue anoxia. Hotchkiss [42] does not feel that the eosinophil count is dependable, and so the controversy goes on. I have nothing of value to add to the subjects discussed in the foregoing paragraph and am not prepared to add to the knowledge already accumulated concerning fluid composition and cellular elements. It is hoped, however, that from all the experimental and clinical investigation now being carried out on the subject, new light will eventually be shed on it to benefit the patient, who, in the final analysis, is the one who should benefit most as medicine progresses.

Samuels,[26] in one of the few references I could find dealing strictly with otitis media in children, reported a series of 116 adenotonsillectomies, of which 14 patients, or a little over 12%, were treated at the same time for secretory otitis. Ages of the children ranged from 18 months to 10 years, and treatment consisted of myringotomy and spot suction. Of the 372 patients reported by Suehs,[17] 120 were children children under 10. Most authors on the subject point out the fact that secretory otitis is common in children, and some say it is most prevalent in children. The now well-known symptoms of hearing loss, "stopped-up" feeling in the ear, and tinnitus have been given attention throughout the literature. However, these symptoms are either absent or not reliable in young children.

This study was prompted by what I felt was a marked increase in the number of young children I was seeing with fluid ears. Fifty consecutive surgical cases of adenoidectomy and/or tonsillectomy were reviewed, and I found to my amazement that no less than 12 of these patients had secretory otitis and that 9 of them were bilateral. Of the three unilateral cases, one child had a chronic perforation of the other drum, but a dry ear. These figures represent an incidence of 24% in a group of young patients referred primarily for evaluation of their tonsils and adenoids because of the usual upper respiratory symptoms associated with infected or enlarged tonsils and/or adenoids. Moreover, and of great importance, the majority of the patients were referred to my care by other physicians; yet the fact that 24% of them had otic pathology was unknown either to the physician or to the parents. Of the 12 patients with secretory otitis, 10 were 5 years of age or under, the youngest being 27 months. One child was 8 years of age, but her mental age was 3; the 12th patient was aged 10. This was the child with one perforated drum of which her parents had knowledge. The hearing in that ear was almost normal, but the parents were not aware of the fact that the opposite ear had a hearing loss of almost 20 db. Of these 12 children, then, not one came for consultation because of ear complaints, except that, on questioning, several of the parents stated their children had frequent earaches, for which the family doctor or pediatrician often gave a "shot" of penicillin, usually followed by relief of the symptom, and the patient was pronounced "cured."

In addition to the above 12 cases, 25 additional cases with secretory otitis media seen in the past year were reviewed. Of these only eight were unilateral. Of this group 13 were 5 and under, and 9 more were in the 6- to 7-year group. The remaining three children were 12, 13, and 14. It is noteworthy that most cases of the disease in children are bilateral.

Considering the combined 37 cases together, presenting complaints in order of frequency and importance were frequent earaches, loss of hearing, mouth breathing, frequent severe and prolonged sore throats, and lastly, nasal speech. Those patients aged 6 years and over usually presented themselves with hearing loss, noted either by themselves, their parents, their teachers, or, rarely, a loss noted in the school hearing test program. In the younger children help

was sought because of frequent earaches, but only rarely was hearing loss mentioned.

The inciting etiologic factor was difficult to determine, as was time of onset. That fact can be readily understood when one realizes the difficulties encountered in working with children who don't even know they have a hearing problem. In the older children who were aware of their difficulty, the duration ranged from a few days to several years. The commonest inciting factor, where known, was an upper respiratory infection, with or without ear pain, and followed by hearing loss or stuffiness of the ear.

Diagnosis is usually not difficult if one keeps the disease in mind in all young patients, not just in those with ear complaints. As in all phases of medicine, a good history and a thorough and complete physical examination are essential. Fortunately the tympanic membrane is most frequently translucent in children, and the familiar amber discoloration can readily be seen. Very often the typical chalky whiteness of the malleus handle is also evident, particularly in ears where the process has been present for considerable time. As has been repeated so often, a fluid level is rarely seen since the tympanum is invariably completely filled. Hotchkiss [42] and Davison [43] have described the decreased motility of the drum when using the pneumatic otoscope. So far as I am concerned, the altered motility of the tympanic membrane under examination with the pneumatic otoscope is the greatest single aid to diagnosis of secretory otitis media in children that I know of. In nearly all instances of fluid in the middle ear the motility of the drum membrane is reduced to a more or less degree, or, if it is not reduced, motion is extremely sluggish. In older children inflation and auscultation are sometimes possible, but I have largely discarded that method of demonstrating fluid in favor of the pneumatic otoscope, which is not in the least painful, frightening, or unpleasant to a small child. The use of the otoscope gives the added advantage of magnification as urged by Jones.[21] In this connection the pneumatic otoscope modified for a manometer as described by Moose [44] might be helpful in establishing a diagnosis. The binaural transmission test used by Armstrong [45] could also be of value, although I confess I have tried neither of those methods.

As would be expected, hearing was decreased in each case. Audiograms were obtained on all the older children, and demonstrated hearing losses generally from 10 to 20 db. In a few cases the loss was as high as 30 db. Usually the loss was somewhat greater for high tones, in contrast to the hearing curve of patients with a loss due to Eustachian tube obstruction but without fluid, where little or no loss for high tones was shown. In the younger children the whispered voice test was most satisfactory, and with the parent closing one of the child's ears, the hearing loss was often dramatically demonstrated, since the parent could hear the whispered words very clearly when the child could not.

While on the subject of hearing loss from secretory otitis, the school hearing conservation program deserves mention. Suehs [17] pointed out that many of the children in his series were seen because of impaired hearing discovered during school hearing surveys. I have seen relatively few cases from that source, and I feel perhaps many are going undetected. Certainly the value of school hearing tests cannot be denied, but unfortunately children being tested may have a hearing loss of 15 to 20 db. for pure tones and yet not be suspected of having ear pathology. Familiarity with the work of Newhart and Reger [46] will readily demonstrate the problem involved.

Regarding treatment, the patients in the first group who underwent adenoidectomy or adenotonsillectomy were subjected to myringotomy and spot suction at the same time, under general anesthesia. Each patient recovered promptly and completely from his ear disease and hearing loss. Comparison with another series in a previous year, when catheter inflation was also performed,

showed recovery to be more prompt in the group not catheterized. Hotchkiss,[42] Suehs,[17] and others have had the same experience.

Of the second group of 25 children, all were initially treated conservatively with vasoconstrictors and antihistamines for periods of two to four weeks. Eleven patients responded to medical management, but the remainder required myringotomy. All but one are considered permanently cured. The one child not cured has a malignant brain tumor that has been incompletely removed, and that is believed to be the basic cause of his ear problem. Four of the eleven patients treated conservatively had previous adenoidectomies, and had no recurrence of secretory otitis. However, the remaining seven had moderately large adenoids, had recurrence of their fluid ears, and were treated by adenoidectomy and myringotomy, resulting in permanent cure. The consistency of the fluid obtained from these ears by myringotomy varied from serous to viscid mucous, with the distribution almost equally divided. Smears and cultures were made from a number of them, but no organisms could be demonstrated. Before and after audiograms are superfluous and space-consuming, and hence are not being shown.

Two very important features of the second group must be pointed out, and further discussion of those features will be taken up later. Five of the children had been treated for their hearing loss elsewhere by adenoidectomy or adenotonsillectomy, but the diagnosis of secretory otitis had not been made. In fact in two instances, according to the parent, the physician had overlooked examining the ears. In one of those children, and four additional ones in the group, previous adenoidectomy had been done, but a large adenoid growth was nevertheless present. Surgery had been done so recently in these cases that it is doubtful the tissue was due to regrowth or hyperplasia. Three typical histories demonstrate the problems involved in the cases under discussion.

Case 1.—Boy, age 5 years. His mother stated that he had a nasal sound to his voice, and that she had noticed that his tonsils were very large. Examination revealed very large tonsils with many palpable cervical nodes, and adenoids so large they almost completely obstructed the nasopharynx. Both tympanic membranes showed the typical amber discoloration and chalky white appearance of the malleus associated with fluid in the middle ear. Motility of both drums was markedly decreased when examined with the pneumatic otoscope. Hearing was reduced to 8/15 for whispered voice in both ears. Treatment was adenotonsillectomy and bilateral myringotomy with aspiration of thick mucus from each middle ear. Recovery and return of hearing to 15/15 for whispered voice was prompt. There has been no recurrence nine months later.

Case 2.—Boy, age 4 years. This boy had been subject to repeated earaches bilaterally, often accompanied by discharge, for two years. These attacks were always treated with penicillin. One year previous to examination his tonsils and adenoids had been removed, but there was no change in the recurring attacks of otitis media. Now his pediatrician wanted to put him on daily doses of chlortetracycline (Aureomycin) for the winter. Examination revealed the typical picture of secretory otitis media described in the previous case. There was a moderately large residual adenoid growth. Bilateral myringotomy was performed with adenoidectomy. Thick tenacious mucus was aspirated from both middle ears. Ten months later there has been no recurrence.

Case 3.—A 14-year-old boy with a history of having been hard of hearing for three years and of having repeated ear "abscesses" for seven years. Adenotonsillectomy had been performed one year previously, and adenoidectomy repeated two months later, with no improvement in hearing. Examination revealed the presence of a very large adenoid growth. Both drums showed a bluish tinge, and motility was decreased. Average hearing loss was 26 db. Adenoidectomy was repeated, and bilateral myringotomy released thick serosanguineous fluid from both ears by aspiration. Hearing returned to normal and has been so for 11 months.

In all instances myringotomy was done under general anesthesia. Jones,[11] Hoople,[16] and others have reported the relatively little discomfort involved when the noninflammed tympanic membrane is incised without anesthesia. I am well aware of that fact in dealing with adult patients and from my experience with my own case of secretory otitis. However, in handling all but the most mature children, the confidence that

has so often been painstakingly built up in the child can be destroyed in an instant if he is frightened or emotionally upset by the thought of being hurt.

My review of the literature and of my own experience with secretory otitis media suggest a number of comments. Most authorities agree that the disease is increasing in its incidence. Moreover, the incidence of its occurrence appears to be much greater in children than in adults. In a recent series of 1016 affected ears reported by Armstrong,[45] 793, or 78%, occurred in children. On the other hand, the higher incidence in children has been noted since the last century.

Although considerable controversy still exists concerning underlying causes, the basic etiology in children is generally agreed to be Eustachian tube obstruction. Although adenoid enlargement and hypertrophy of nasopharyngeal lymphoid tissue are mainly responsible, variations in the size of the pharyngeal opening of the Eustachian tube, as pointed out by Tremble,[47] must be taken into consideration. Bezold[4] discussed in his lectures the rarefaction of air in the middle ear caused by tubal occlusion, and the resulting "hyperemia ex vacuo" which resulted and caused a transudation of fluid into the middle ear. Since that time the controversy over transudate and exudate has not been resolved.

The judicious use of antibiotics for the treatment of certain upper respiratory and ear infections is commendable, but the consequences of unwise and all too often promiscuous use and misuse of these drugs, as pointed out by Schenck[18] and other authorities, is well illustrated in the two series of patients presented. Nor is the attending physician always entirely to blame in many instances, for all too often the person concerned well nigh insists that he or his child be given a "shot" of penicillin for his illness. In cases of otitis media antibiotics should be administered only where there is unequivocal indication for their use, and in every instance repeated examination of the ear drum and hearing must be done to insure complete recovery.

In spite of the attention received in recent literature, the diagnosis of secretory otitis is all too often missed. Hoople,[16] Rawlins,[48] Singleton,[34] and many others have called attention to that fact. Some writers feel that the disease has not received the attention it deserves, while others are of the opinion that it is receiving more of the consideration it merits, not only from otologists, but from the pediatricians and general practitioners as well. I share the view of the former group and believe, with Armstrong,[45] that we as otologists must share the blame. We must not only train the men in our specialty, but it becomes paramount that we must acquaint the rest of the profession with the prevalence as well as the seriousness of secretory otitis media. This task can probably best be accomplished by presenting discussions concerning the disease to the profession at large and by active participation in local and state medical society meetings.

Diagnosis is not difficult in children if the condition is always kept in mind. It must be remembered that many young children do not complain of ear symptoms, and often their parents do not suspect a hearing loss. Frequently a child accused of inattention or poor progress in school is suffering from hearing loss due to secretory otitis.

Myringotomy, under general anesthesia, is the treatment of choice in young children. Conservative measures may be tried for a brief interval. They are most successful in cases of the disease which follow an attack of acute otitis media. Hilger[49] suggests, particularly in the care of patients from outlying areas, self-inflation with a small ear syringe used as a Politzer bag, and repeated testing of the child's hearing by the parent using the whispered voice technique. However, there should be no temporizing with conservatism if recovery is not prompt. Politzer[50] and Bezold[4] advocated myringotomy for fluid in the middle ear, just as many authorities have subsequently. Nevertheless, in spite of the many reports in the literature advocating myringotomy for this condition, in spite of the frequent reports concerning

the almost complete absence of complication from myringotomy, many otologists remain reluctant to utilize this form of treatment.

Before I leave the subject of myringotomy, the fact that the presenting complaint of many of these children was frequent earaches, for which they had been treated with antibiotics, deserves comment. I am not assuming that they were improperly treated, but surely myringotomy must have been indicated for some of those cases of acute otitis media. Schenck [18] pointed out that many general practitioners and pediatricians hold the erroneous view that myringotomy is obsolete. Harrison,[51] in an interesting discussion, admitted that he began the practice of otology with the same thought, but two years later, after reading of serous effusion and conduction deafness following incompletely resolved purulent otitis media, he began to perform paracentesis on ears that failed to respond quickly to antibiotics. He immediately found that his patients recovered more promptly, with fewer instances of permanently impaired hearing resulting. Woodward [28] wisely suggested that physicians caring for cases of acute otitis media should either be able to perform a competent myringotomy or should refer the child promptly to an otologist when the operation is indicated. Furthermore, in order to implement our hearing conservation programs, hearing tests should be done after recovery from otitis media, if not by audiometry then at least by whispered voice technique.

Samuels [26] pointed out that secretory otitis commonly appears in children in whom the adenoids have not been removed. King [52] presented a series of 56 cases of fluid in the middle ear in children with tonsils and adenoids. It is interesting to note that the great majority of children presented in this series had large adenoids. Lindsay [53] has spoken of the swing of the pendulum toward extreme conservatism in recommending removal of tonsils and adenoids. Suddenly, then, in a discussion concerning secretory otitis in children, another controversy confronts us. Proud [54] feels adenoidectomy may bring relief in persistent cases of the disease and advocates myringotomy at the same time. Gale [55] and others recommend removal of adenoids at any age if they are causing Eustachian obstruction, while other writers feel that adenotonsillectomy is of no benefit to a child, though it must be kept in mind that they are not always specifically discussing treatment of secretory otitis. Blassingame [56] hoped for more realism in tonsil and adenoid surgery and noted the importance of thorough removal of the pharyngeal tonsil. He felt that at least 50% of the cases are operated upon by persons not qualified by training to perform the operation. Unfortunately, from this study, it would appear that some men who are considered qualified are not as thorough as they should be. At any rate, in most cases it appears that secretory otitis media is a definite indication for adenoidectomy in children.

It is a well-known fact that where there are several known methods of therapy for a disease, no one treatment approaches 100% of success. So it is with irradiation of the nasopharynx following adenoidectomy, another area of controversy. Within that controversy another exists, whether to irradiate by the method of Crowe, or with x-ray therapy. The arguments pro and con are well known, and, since they seem to go beyond the scope of this paper, they will not be discussed here. My experience with irradiation has been much the same as that of Meltzer [57] and Day,[58] and I feel that it has no place in the treatment of secretory otitis and nasopharyngeal lymphoid tissue in children, except in those doubtful cases where lymphoid tissue continues to recur. I am in full accord with Baron,[59] who stated in a recent paper that the most effective treatment of nasopharyngeal lymphoid tissue is meticulous surgery, which has no therapeutic equal.

I shall be the first to point out that I was extremely fortunate with this series in that all but one case recovered promptly. I have not had that good fortune with all previous series, although it has always been my

experience that a much larger percentage of children recover from secretory otitis more promptly than is the case with adults. King[52] found in his series of 56 children that serous ears healed more readily than mucous ears. That finding is in accord with the work of Politzer,[2] Ivstam,[15] Hoople,[16] and others. In the treatment of chronic cases, Rawlins[48] and Jones[21] have advocated self-inflation after myringotomy. Suehs[60] and Armstrong[61] suggest the use of plastic tubing drains placed through the incision in the drum.

It must be remembered that secretory otitis media is not only a disease of the middle ear, but of the mastoid as well, as pointed out by Singleton.[34] Bezold[4] described the fluid running back into the mastoid with change of position. This fact is readily appreciated by anyone when, after myringotomy and evacuation of fluid, the tympanum refills again within a few minutes. Jervey,[62] Tobey,[63] and Cody[64] have reported cases requiring surgery, and Suehs[17] has discussed the indications for mastoidectomy. Several years ago I was confronted with a persistent bilateral case of secretory otitis in an 8-year-old girl. Bilateral simple mastoidectomy was finally necessary before recovery was effected.

The possibility of irreversible changes taking place in the middle ear and leading to permanent deafness in cases of neglected or chronic secretory otitis must not be overlooked. These changes have been pointed out by Politzer,[2] Woodward,[28] Shambaugh and Derlacki,[39] and others. They occur as adhesions, and, in some cases, acquired cholesteatoma. I have had experience with the latter on two occasions. In one instance a 10-year-old girl had developed attic cholesteatoma when seen two years after an attack of secretory otitis. Modified radical mastoidectomy was necessary. In the other instance an adult was found to have developed cholesteatoma some three years after having secretory otitis. In this respect the findings of Simpson[65] are quite remarkable. In his series of cases of chronic otitis media, serum cholesterol levels were high in all patients with cholesteatoma. He believes that an effusion into the attic of serum high in cholesterol may initiate attic cholesteatoma. This finding may support the view of those who feel that hypothyroidism can be a cause of secretory otitis.

Summary and Conclusions

The available literature concerning secretory otitis media has been reviewed, particularly in regard to the disease in children.

My series of 37 cases has been presented, with emphasis on diagnosis and treatment.

The incidence of the disease is increasing, notably as a result of frequent and indiscriminate use of antibiotics, and secondarily because of extreme conservatism in recommending adenoidectomy in children. Increased vigilance is also a factor.

There is evidence that further education of the medical profession is necessary in order that case finding be improved and that diagnosis of the condition not be overlooked.

Secretory otitis media should be suspected in children who have frequent earaches or a hearing loss. It is usually bilateral in children.

Secretory otitis media is a serious disease, and its complications can be far-reaching in their social and economic effects upon the patient.

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Secretory Otitis Media and Nasal Allergy

Preliminary Report of Sixty-Six Patients Treated with Promethazine

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Nasal allergy as a complicating and often confusing accompaniment to diseases of the upper respiratory system has been frequently reported in medical literature. Silbert,[1] in stressing the necessity for differential diagnosis of this syndrome, estimates that 75% of the patients he sees exhibit some form of sensitivity; and Gehrand,[2] in a clinical study of 134 cases selected for extreme susceptibility to respiratory infections, found well-defined clinical evidence of allergy in 74%.

Promethazine,* a potent and long-acting antihistamine, has proved effective in a wide variety of allergic upper respiratory disorders.[3-6] The therapeutic efficiency of this drug, combined with its freedom from undesirable side-effects, suggested a clinical evaluation of promethazine for symptomatic relief of secretory otitis media and nasal allergy concurrent with specific treatment of the underlying condition.

Method

The study comprised 66 patients (61 adults and 5 children) ranging in age from 8 to 73 years, who were seen in private practice. Of these, 27 were suffering from secretory otitis media, 27 from perennial allergic rhinitis, and 12 from seasonal rhinitis. Bacterial infections were a complication in six patients with perennial rhinitis; deviated nasal septum was present in eight, and nasal polyps in two cases. Twenty-two patients had used antihistamines previously, and received little benefit or experienced undesirable side-effects.

A definite effort was made to determine whether the symptoms in each case were caused primarily by a bacterial infection, allergy, or a combination of both. If there was clinical doubt as to the origin of the disorders, the nasal secretions were cultured to demonstrate the presence of pathogens. Eosinophil counts were performed repeatedly; however, they appeared to have little significance in this study. In most of the cases of secretory otitis media, the count was 0 or 1; in cases of definite or probable allergic origin, counts ranged from 0 to 34%, the maximum occurring during the pollen season. Staphylococcus aureus, the only pathogen demonstrated, appeared in the cultures for 10 patients.

Antihistamine medication consisted of a daily oral dose of 25 mg. promethazine at bedtime in order to take advantage of the definite soporific action of the compound. In five cases, the dosage was reduced to 12.5 mg. without lessening the effectiveness of therapy, when the sedative effects, desirable during hours of sleep, extended into the following day.

Vasoconstrictors were administered topically according to individual indications as a supplement to treatment of local symptoms. Systemic medication included the use of sulfonamides and various antibiotics for control of the associated infection. In the case of three patients suffering from seasonal rhinitis, hyposensitization was attempted. Patients with secretory otitis media were instructed in the Valsalva maneuver. Treatment was continued from one week to four years.

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* Phenergan, promethazine hydrochloride, was supplied by Wyeth Laboratories.

Results

Promethazine proved a highly successful adjunct to therapy in nearly all cases, as evaluated on the basis of subjective response and objective evidence: 88% of the patients considered themselves symptom-free or definitely relieved. Nine of the ten with staphylococcal infection reported that they were entirely well or that they had experienced significant improvement. Under clinical examination, 84% were obviously free of symptoms or markedly improved. In those who responded to treatment, significant change was detected, subjectively and objectively, in three days to three weeks.

Secretory Otitis Media.—Results in these cases indicated definite success in treatment. Of 27 patients 25 (92%) were improved or symptom-free, and of these, 13 (48%) were diagnosed as clinically well after continuation of therapy for five days to three weeks.

One patient in this group had suffered bilateral otitis media accompanied by hissing type tinnitus, with retraction of the ear drums, over a period of six months. After three days' treatment with 25 mg. of promethazine each night, a vasoconstrictor, and use of the Valsalva maneuver, all symptoms subsided. In another case, bilateral secretory otitis media, of three months' duration, had not responded to Eustachian tube catheterization, use of tripelennamine hydrochloride, and adenoidectomy. After 10 days' medication with promethazine, phenylephrine hydrochloride, and use of the Valsalva maneuver, the fluid had entirely disappeared from the middle ears and the air conduction became better than bone conduction bilaterally. The patient at the time of this writing had remained asymptomatic for three months.

Only two patients in this group failed to respond to therapy including promethazine medication. One of these reported subjective improvement after medication with one 25 mg. tablet per day for one week, although there was no objective clinical change. The second patient, after three weeks of medication, required roentgen therapy to the nasopharynx.

Myringotomy was not required for any of the 27 patients with otitis media.

Perennial Allergic Rhinitis.—Of the 27 patients treated for perennial allergic rhinitis, 85% experienced subjective benefit: 15 reported that they were symptom-free, and 8 felt improved after treatment. Clinical improvement was determined objectively in 78%.

One patient with septal deviation and partial obstruction complicated by a phenylephrine reaction showed such improvement that plans for a submucous resection were canceled. Another patient reported that her symptoms were not only relieved by promethazine medication, but that she is now able to eat many foods which had previously provoked allergic reactions. One patient who had undergone a polypectomy experiences a return of nasal congestion when medication is omitted for one night. A fourth patient, subject to frequent upper respiratory infections, has remained symptom-free for a year on nightly doses of promethazine by mouth.

Of the six patients in whom no clinical change occurred, one had a primary diagnosis of hypertrophic mucosa with nasal obstruction, and in another, nasal polyps were present. Surgical measures were required, but promethazine medication was continued for these patients since it afforded some symptomatic relief. Two patients had previously failed to respond to therapy with a number of other antihistamine preparations and another suffered from postnasal drip complicated by a staphylococcal infection. One patient neglected to follow instructions for treatment.

Seasonal Allergic Rhinitis.—Subjective relief occurred in 75% of the 12 patients suffering from seasonal allergic rhinitis, with 7 becoming symptom-free. Clinical improvement was noted objectively in 75%.

One patient who had previously experienced only temporary relief with cortisone nose drops remained completely symptom-

free on a daily dose of 25 mg. of promethazine during the pollen season. Another experienced complete relief on promethazine medication after attempts at hyposensitization had been ineffective.

Side-Effects

A 25 mg. dose of promethazine taken at bedtime produced no untoward effects in 51 of the 66 patients (77%) included in this study. The only side-effect reported was daytime drowsiness, which was controlled in two cases with amphetamine, 5 mg. in the morning and at noon, and which disappeared spontaneously after the first few days of medication in four cases. One patient who was drowsy for the first four days did not again experience this reaction during five months' continuous treatment. Five patients obtained relief without loss of therapeutic benefit when the dose was reduced to 12.5 mg.

No evidence of toxicity was noted, even though treatment in some instances had been continued for one, two, and three years. One patient in the group has used promethazine nightly as a sedative for over four years. She has been under frequent observation because of mild arthritis, and at no time has there been any evidence of depression of the blood-forming mechanism.

Summary

A group of 66 patients (Table) suffering from secretory otitis media, seasonal and nonseasonal allergic rhinitis, and various other disorders of the upper respiratory system received promethazine for symptomatic relief in conjunction with specific therapy for underlying conditions. The usual dosage was 25 mg. taken orally at bedtime. The single bedtime dose of promethazine provided a desirable sedation. The prolonged antihistaminic action carried over throughout the following day, obviating the need of daytime doses, which are usually necessary with other antihistamines and which cause an undesirable daytime drowsiness.

At termination of therapy, 88% of the patients reported definite subjective improvement or elimination of symptoms. In 84% the improvement they claimed could be demonstrated objectively. No serious side-effects were encountered. In the few cases in which the desired soporific effect of the compound was prolonged beyond the hours of sleep, upper respiratory symptoms were controlled by reducing the dosage to 12.5 mg. without loss of therapeutic efficiency. In two instances, control was achieved by the use of 5 mg. amphetamine in the morning and at noon.

Promethazine is considered a valuable adjunct to the treatment of secretory otitis media, and is recommended for symptomatic relief in upper respiratory disorders of established or probable allergic origin.

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Summary of Cases

Disease	Number of Patients	Therapeutic Response: Subjective: No Change	Subjective: Improved	Subjective: Symptom-Free	Objective: No Change	Objective: Improved	Objective: Clinically Well	Improved or Well According to Subjective and Objective Criteria: No.	Improved or Well According to Subjective and Objective Criteria: Per Cent
otitis media secretory	27	1	6	20	2	12	13	25	93
Perennial allergic rhinitis	27	4	8	15	6	19	2	18	67
Seasonal allergic rhinitis	12	3	2	7	3	9	0	8	67
Total	66	8	16	42	11	40	15	51	77
Per Cent		12	24	64	16	61	23		

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The Education of a Young Deaf Child Begins With His Parents in His Home

MRS. SPENCER TRACY, Los Angeles

There is at present no known cure for nerve deafness, and the only treatment lies in education. Now, there sometimes is a tendency to think of education as something that begins more or less automatically at the age of 5 or 6 in the schoolroom and under the supervision of a trained teacher. While formal education might be said to start with school, most of us know, even if we sometimes forget, that learning, all learning for each and every one of us, begins at birth, and if we reflect a little we may even accept the idea that during our first five or six years—that is, during our preschool years—we learn more than in any other five- or six-year period in our lives. Continuously, during these years we are acquiring a mass of information, collecting a vast number of facts, inevitably some fancies, establishing attitudes, some of which we will carry throughout our lives, and developing personalities and a pattern of living which can be modified later only with difficulty. During this period we cannot say, "We will learn," or "We will not learn." We learn. What we learn, how we develop, depends primarily upon our environment and upon the people in it, that is, during our early months and years, upon our parents, and not only upon their knowledge and wisdom but upon their attitudes toward life and toward us, upon their acceptance of us no matter what our problems or handicaps may be, upon their love, their support, and their understanding.

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Communication is, of course, the major handicap of deafness, and communication with our fellow men, it seems to me, is possibly the greatest single factor in living. The degree to which we can understand the spoken or written words of others and the extent to which we can express ourselves, clearly, interestingly, persuasively, often determines overwhelmingly our successes and our failures. Acquiring a means of communication is part of the learning process and one of the principal achievements of a preschool hearing child. Through association and imitation of the sound patterns to which he is exposed, he learns—he *absorbs*—the speech and language which hearing people take so for granted, together with the ideas and attitudes they indicate; and if he enters school with good speech and language—good 5- or 6-year-old speech and language—he does so because he has been exposed to good speech and language in his home. Furthermore, he possesses the tools for the best education of which his mind is capable. However, let us always remember, his ability to use these tools and to take full advantage of his intellectual capacity will always depend in some measure upon his freedom to do so, upon his security, which stems from that acceptance, love, support, and understanding I spoke of.

A deaf baby cannot come by communication and the great reservoir of knowledge dependent on it in the natural and comparatively effortless way a hearing baby does. He must be taught. Through the infinitely slower, more difficult, and never quite adequate or completely satisfying means of sight, touch, and whatever residual hearing he may possess, he can be taught first that

the movements of people's lips carry messages and gradually he can learn to interpret their meaning. This we call lipreading or speechreading. Later he can be taught not only to understand words but also to speak them. If single words were all, the task might be comparatively easy. But he must be taught language—that orderly arrangement of words which conveys meaning—that infinite variety of word combinations, the idiomatic expression which a hearing 6-year-old uses so freely. Language is the great problem, for regardless of the *means*—speech, lipreading, reading, writing, even finger spelling—a knowledge of language, the material out of which all communication is made, is essential. The education of a deaf child truly presents some of the most difficult problems in the entire field of education, and clearly demands—among other things—trained and experienced teachers of the highest quality.

But must this little deaf baby mark time during his preschool years, during those great imitative and habit-forming years, during the speech and language years from which a hearing child can profit so magnificently? Must his education wait for the teacher and the formal program in the particular special school or classroom available to him? Much of it unquestionably must wait. But it must be clear that a deaf baby, too, learns from birth and can use these early years to advantage and for what we might call "a running start."

It is obvious it seems to me that if we are to use these early years we can do so only with the understanding and through the participation of the parents. If we are to help little deaf babies we must begin by helping their parents and as early as possible. How early is possible? In terms of discovering a severe hearing loss, I will cite an example.

Prof. and Mrs. A. W. G. Ewing of the University of Manchester at Manchester, England, tell of a 4-week-old baby up for adoption who was brought to them for a hearing test. The Ewings said that at the time they had never tested so young a child and were doubtful of being able to do so. However, the people insisted, and the Ewings finally gave their opinion, although they were loath to do so, that the infant did have a hearing loss; that is, that he did not react normally to sound. He was not taken for adoption, and it developed later that he was indeed deaf.

So one month clearly must represent "possible." On the basis of much authentic evidence, it is my belief that with awareness, intelligent observation, and the use of simple startle tests, even in the home, a severe hearing loss, uncomplicated by other problems, in most instances can be detected by the age of 6 months. It may still be a number of months and possibly several years before the exact degree of deafness can be determined and an audiogram can be made. The point I am trying to make is that "possible" is much earlier than it is commonly thought to be, and guidance for parents and training for the child need not wait for an audiogram.

"But," someone of you may ask, "supposing what appears to be a severe hearing loss is discovered at such an early age, what can parents actually do to help a child when he is still so young?" I will presume when you say *do* you mean specifically in the way of training for speechreading and speech, because, as I indicated earlier, *everything* parents do—with their child, to their child, in front of their child—affects in some manner and in some degree contributes to not only his learning, but, indeed, sometimes his very ability to learn and the development of his personality, his security, his independence, his ability to relate to people and to communicate with them. However, even at the age of a few months parents *can* begin the special training, *can* lay the foundation for lipreading, language, and speech, for communication in this hearing world, and for a rich, productive and satisfying life. Indeed, there are things they can do with their baby at from 3 to 7 or 8 months that can never be done as easily or in the same way at a later time. Mrs. Irene Ewing, who,

together with her husband, has done so much work not only in the field of deafness generally but of the very young deaf child, has pointed out that this is the period when the baby enjoys being held in his mother's or his father's arms, when he is interested particularly in them and when he watches their faces and reacts to almost each fleeting expression. Later, possibly at 9 to 10 months, he will become increasingly interested in things, in creeping and climbing and in exploration. His attention and interest in people's lip movements will be much harder to gain. During the earlier period attention and interest are there. Also his position in mother's arms produces the shortest practical distance between his eyes and her mouth, and it is not difficult to capture his little hand and lay it against her cheek or throat where he can feel the sound vibrations of her voice. Besides, just being close to her body, he can often be aware of these vibrations.

During this period, hearing children are beginning to recognize the patterns of sound that make up speech and to match them to the appropriate object, action, or idea—"Here comes Daddy." "It's time for your bath." "No, no." "Wave your hand bye-bye." "Patty cake, patty cake." Et cetera, et cetera. Although learning to understand words and language through the recognition of patterns of lip movements is, as I said earlier, infinitely slower and more difficult, principally because there is no lip movement for about 60% of all speech sounds and the patterns are incomplete, still the need for communication is so great within each one of us, if we can't get it in one way, we'll get it in another.

I know of a baby who began to lipread before he was a year old. There is a home movie film which shows his mother plainly saying, "Where are your eyes?" and the baby pointing toward his eyes; and the mother saying, "Show me your nose. Where's your nose?" and the baby pointing to his nose. Fantastic? Yes, it may seem so. But there is a somewhat enlightening story behind this phenomenon. The mother and father, you see, are both deaf. So they were aware that they might have a deaf child. By the time he was 3 months they were quite sure their baby could not hear. Were they shocked and miserably unhappy? Did the mother have a nervous breakdown? No. He was just like they were. They were getting along all right. They were happy. He could be, too. So there was no frustration, no insecurity, and no unhappiness to communicate to the child; instead, complete acceptance, love, support, and understanding. Lastly, they had gone to a good oral school. They knew what to do. They knew that first must come understanding through lipreading, and that in order to learn to lipread the baby must be given an opportunity. He must be talked to, and so they *talked, talked, talked.* I can just see that mother (illustrate)—"Where is your nose?" "There's your nose." "Here's Mother's nose." "Show me your eyes." (Taking his hand and pointing to his eyes.) "There are your eyes."

Here we have the three necessary steps:
Awareness leading to early discovery
Acceptance
A knowledge of what to do

Sometimes the order of the last two is reversed. Undoubtedly, some knowledge of what to do and what can *be* accomplished helps to bring about acceptance.

Although I feel that considerable progress has been made in the last 10 to 15 years, there is still little public awareness, and even less understanding, of the problems of deafness and what can be done about them. There is a tendency I find to think about deafness generally in terms of—shall we say—grandfather who lost his hearing lately. Now grandfather unquestionably is deaf and he has his problems as have, possibly, the people who live with grandfather, but these problems are very different from those of the person who was born without hearing—different not only in degree but in *kind.* A large slice of the public still appears to believe that a child who is born deaf is also born dumb or mute. There are quite a few people, I have discovered, who

are surprised to learn that deaf children have vocal cords. References to "deaf-mutes" and the even more unpalatable term, "deaf-and-dumb," and the labeling of schools as institutions for deaf-mutes as was once the case in the United States and is still the custom in some countries, all serve to perpetuate this misconception. There is little awareness that thousands of babies are born without hearing from causes as yet unknown and that perfectly healthy parents with no history of deafness in the family of either one can produce a deaf child.

Our experience at the Clinic leads us to the belief that very few parents—or doctors—check a baby during his early weeks or months for the always possible hearing loss. A hearing loss cannot be seen and it is not expected, as it was in the case of the young deaf couple, nor even usually thought of. Discovery comes, as a rule, only through what we might call an incident or when at the age of 2 or 2½ years the child has not yet commenced to talk. Even then, in our experience with nearly 9000 young children and their families spread throughout most of the world, we find that more often than not parents have a hard time, first, in getting from their doctor a confirmation of their own suspicions and, second, in getting help—even information on where to go, or to whom to write, for help. Possibly part of the problem is that many medical people, not knowing how much can be done for a deaf baby educationally, cannot realize the pressing need for early diagnosis. There is, we find, often a considerable lapse of time not only between that first gnawing suspicion that "something is wrong" and the finding of help, but between the diagnosis and the information, which, as numbers of parents have phrased it, has "opened new worlds" to them and their children. The greater this lapse, the harder it is, generally, to help the parents. Help should come instantly—almost in the same breath with diagnosis, and it should begin with the acceptance of the parents and their feelings. Our psychologist often says it is of no help to tell parents there are worse things than deafness. At that moment, to them there is nothing worse. Adults, like children, need acceptance where they are. Any real progress can only begin from that point. It's of no use to say you *should* be over here, if you are actually over there. Parents need help desperately—not only as parents but as people. So, at John Tracy Clinic we are concerned not only with facts but with feelings, because sometimes parents cannot use the information we are able to give them because of their feelings. Indeed, sometimes they cannot even hear it.

So, although I believe the Clinic is often thought of as a school for young deaf children, it is really a parent center, where parents of deaf infants and young children may come or may write for encouragement, information, guidance, and training. To be sure, we do have a nursery school for a limited number of children, 2 to 5 years of age, but this is only one phase of the total parent program and no child enters the nursery school without his parents. The family is enrolled as a unit—the parents go to school, too.

The total program of the Clinic includes consultations, clinics, nursery school, a six weeks' summer session, a correspondence course, counseling, a teacher-training course in association with the University of Southern California, and an organized program of research. At the initial consultation both mother and father are asked to be present with their child. If they were not referred by an otologist, we urge that the child be seen by an otologist before this consultation, but we do not demand it; however, if later the family is to be enrolled in the nursery school, and before a child is fitted with a hearing aid, we do insist upon an otological examination. This first consultation is aimed not only at learning what we can of the extent of the child's hearing loss but at giving encouragement and information to the parents and at finding out all we can about the total family situation, so that we can offer the best guidance and plan the best program possible for *them*. A series of 10 weekly clinic days gives further op-

portunity to study the child and to orient the mother, preparing her for her "job" and for the classes available not only to her husband and her but to all adult members of the family who wish to come. There are at present nine regular weekly classes open to all parents of deaf children. These have to do with, first, the growth and development of any child and how the attitudes and actions of parents affect that growth and development, and, second, with the problems of deafness generally and how families can help their children in learning to lipread and in acquiring language and speech. There are also two classes on different levels which are pure group therapy.

Deaf children have all of the needs of hearing children, so the nursery school, which I already have mentioned, is set up as any good nursery school for hearing children would be, with trained nursery school teachers in charge. To this program we add daily periods, ranging from five minutes to possibly an hour depending on the age and readiness of the child, during which each youngster is tutored individually by a trained teacher of the deaf so that he may develop at his own rate of speed. The mothers attend one day each week to observe and, when they are able, to participate; also both parents attend at least two of the weekly classes.

The six weeks' summer session, when parents and children living outside Los Angeles community are enrolled, is a sort of concentrated and stepped-up version of the winter nursery school. Parents attend classes and have supervised observation periods in the nursery school and the tutoring rooms daily. We have had families not only from all over the United States but from Mexico, Hawaii, Canada, and Japan, as well.

In line with our belief in the importance of feeling and of help to parents as people, we not only have group therapy but, when wanted, individual psychological counseling.

I spoke of families throughout the world with whom we had been in touch. We have a Correspondence Course available to parents of children between the ages of 2 and 5 years inclusive. Up to the present time, we have had nearly 9000 parents from 52 countries enrolled in this Course. It is sent out in 12 monthly installments, although the material may cover several years of work, as in the case of the 2- to 3-year-olds. This is a service—a course of treatments—all doctors have at their disposal. As with all of our services, there is no charge. The only provisos are that the parents read and write either English or Spanish, the only two languages in which the Course is given, or that they be able to have the installments translated as well as their own monthly reports to the Clinic, and that they will cooperate and work to the best of their ability. Although the Course is not suitable for children under the age of 2 years, I hope by now you do not need assurance that there is information, encouraging, stimulating, and practical, that we are able to send to parents regardless of how young their children may be.

There are many possible ways in which guidance, training, education—call it what you will—can be given to parents; however, any and all early help for both parents and children is dependent upon early discovery, and early discovery to a great extent is dependent upon the awareness of all medical people who at one time or another see infants and young children. It is not, as some appear to think, the sole responsibility of the otologist, but falls, it seems to me, primarily within the province of the pediatrician, the family physician, and the general practitioner. These are the people who first see young children. The otologist seldom enters the picture until someone suspects trouble. But when he does, parents have every right to look for help. This does not mean that every otologist should be expected to know how to teach lipreading and speech, although it does seem to me that some fundamental knowledge on the subject of lipreading might often be useful to those who work with hearing and with people as well as with ears, but he should know that lipreading and speech can be taught. He need

not concern himself with techniques or methods, but he should have at hand information as to where the needed help can be got and what services there are for both parents and children, not only in the community, but within reach of a postage stamp.

To the medical men in other fields I mentioned a moment ago, in the hope that these words may in some way reach a few of them, I would like to say that there is one very simple device that has seemed to me might bring to light some cases of deafness much earlier than usual. Would it not be possible when a baby is first seen, whether he is sick or well, in a clinic, a private office, a hospital or a home, along with the many questions so often asked—"How well does he eat?" "How well does he sleep?" "How well does he go to the bathroom?"—there might be included, "How well does he hear?" Although not completely air tight, I believe it would serve to help create an awareness that sometimes children do not hear well—or at all—and if they don't, the time to find out and to do something about it is *now*.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

A Rapid and Simple Technique for the Detection of Nonorganic Hearing Loss

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The term "hearing loss" denotes an impairment of hearing acuity which is commonly defined clinically as either organic or nonorganic loss. Since World War II, hearing clinics throughout the country have reported an increase in the number of nonorganic losses of psychogenic origin or malingering.

There are many apparent benefits to be derived by persons displaying a handicap in hearing acuity. A few of these might be enumerated as follows: exemption from military service, Veterans' Administration disability pensions, and workmen's compensation due to occupational trauma. Hearing loss as an explanation for abnormal behavior, academic deficiency, or as an "attention-getting" mechanism in children is not unusual. As more is done to compensate and benefit the acoustically handicapped person, hearing losses become more attractive to the potential malingerers and those with psychogenic hearing losses. The responsibility of detecting the person with this nonorganic loss falls to the otologist and/or the audiologist.

Most people consider a hearing loss extremely easy to feign and extremely difficult to disprove. Those persons involved in the measurement, diagnosis, or treatment of auditory deficiencies realize that, given the necessary equipment, the patient's hearing can be accurately measured both qualitatively and quantitatively even without the cooperation of the patient.

Present testing procedures for the detection of nonorganic hearing loss are certainly both adequate and accurate. These include such techniques as the Stenger, the modified Stenger, the Doerfler-Stewart, the swinging-tone or swinging-voice test, the delayed feed-back, the galvanic skin response, the Lombard, and the use of repeated threshold tests. All of these tests are excellent and all make it extremely difficult for the patient with a nonorganic loss to go undetected. Most of these tests, however, require something more than the average commercial clinical audiometer as found in most offices. Various types of special equipment needed for the above tests include speech-reception test equipment, masking-noise generators, a two-channel audiometer, tape recorder, or psychogalvanometer.

It has become virtually impossible to feign a hearing loss when the above series of tests for a nonorganic loss is administered. A problem arises outside the testing clinic when special equipment and the need for these time-consuming techniques are required. These tests are not routinely administered to screen possible nonorganic losses from the numbers of patients displaying organic auditory difficulties. Generally, these procedures are administered only when inconsistencies either in the case history, the routine clinical tests, or the rela-

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This study was completed while the author was Clinical Audiologist at the Speech and Hearing Clinic, University of Oklahoma Medical Center, Oklahoma City.

tionship between the two are noted. Practiced and experienced malingerers can present a very acceptable pure-tone audiogram, which may correlate with speech reception and discrimination scores and will not show discrepancies as related to the case history. For this reason it is possible for a malingerer to pass through yearly examinations completely undetected.

I feel that the technique to be described may be of assistance in overcoming the aforementioned problems. The technique has as its objective a rapid and simple procedure requiring no special accessories beyond the simple basic clinical audiometer to indicate the presence of nonorganic loss or overlay. The procedure can be given as a screening test for the detection of nonorganic losses and to indicate the need, or lack of need, for a further investigation of the hearing loss.

The rationale for the testing procedure is evolved from the premise that the successful malingerer must relate the level of feigned threshold to his true organic threshold or some other loudness level. The purpose is to destroy his perspective of loudness levels and confuse his ability to relate the loudness of the presented stimulus tone at a predetermined level.

The procedure involved in the technique proposed in this paper is simple and can ometric procedure with little additional time be integrated into a routine pure-tone audirequired to complete the testing.

Equipment

The equipment used in this study consisted of an Allison 21-B clinical audiometer, situated in a dual, sound-treated clinical testing room. The equipment is used for routine clinical testing. Any accurate pure-tone audiometer could have been utilized.

Subjects

Three groups of subjects were used in the present study.

The first group consisted of patients seen in the clinic who had been positively diagnosed as cases of nonorganic hearing loss from a battery of the existing tests for the detection of malingering and/or psychogenic loss. Twenty-five nonorganic loss ears were used in the study.

The second group involved 25 normal ears. This group was asked to malinger. Each subject adjusted his own level of the nonorganic loss to be reported. Each of these subjects was acquainted, either through training or experience, with audiometric techniques and would perform with greater success than could an untrained listener.

The third group consisted of 25 ears displaying a true organic loss. This group was used as a control group in the study.

Procedure

Each subject was given a pure-tone audiometric test to determine his air-conduction thresholds at 500, 1000, and 2000 cps. The method of tone presentation was, initially, an ascending series of intensity presentations of the stimulus tone. The tone was randomly interrupted by the examiner as is done in the clinical situation. Each subject was asked to indicate when the tone was heard by raising his finger.

Each ascending series of presentations was begun at an intensity reading of 10 below zero on the audiometer intensity indicator regardless of the threshold level. The intensity was increased in 10 db. steps until the subject responded to the tone. The intensity was then decreased in 5 db. steps until the subject again failed to respond. Once again the intensity was increased in 5 db. steps, and a threshold was established. The threshold was recorded for each of the three frequencies used.

The tones were then presented, using a descending series of intensity presentations. The same three stimulus tones, 500, 1000, and 2000 cps, were initially presented at a level of 90 db. and reduced in 10 db. steps until the subject failed to respond. At this point the intensity was increased in 5 db. steps until the subject again responded. This second threshold was established in a reverse direction from the first noted above. In both phases of the study, the lowest intensity eliciting a response was recorded as the threshold value. Since the smallest attenuation normally used in a clinical situation is 5 db., it was felt that the lowest intensity value would minimize any possible error which might tend to show discrepancies in the two thresholds obtained by the two methods.

Results

The differences in threshold as obtained by the two methods of intensity presentation are shown in the Table.

The diagnosed malingerers displayed a mean difference between the two thresholds

Differences Between Ascending and Descending Thresholds for Four Groups

	Descending Minus Ascending Thresholds		
	Frequency		
	500 cps	1,000 cps	2,000 cps
Diagnosed nonorganics	21.00 db.	24.40 db.	22.04 db.
Diagnosed organics	1.40 db.	0.80 db.	−0.20 db.
Normals, malingering	22.00 db.	29.40 db.	29.20 db.
Normals (previous study)		−2.10 db.	

for the 500, 1000, and 2000 cycle average of 22.20 db. The individual frequencies showed a mean difference of 21.00 db. at 500 cycles, 24.40 db. at 1000 cycles, and 22.04 db. at 2000 cps. In each case, the ascending threshold was lower than the descending threshold.

The group of normal hearing persons asked to malinger showed a mean difference between the two thresholds of 26.76 db. for the average of the three tones. For the individual frequencies, the mean differences were as follows: 22.00 db. at 500 cycles, 29.40 db. at 1000 cycles, and 29.20 db. at 2000 cycles. Again, as in the previous group, this group displayed a lower threshold by use of the ascending series.

In the data for the group of true organic loss ears, the mean difference between the two thresholds was 0.70 for the three-tone average. At 500 cycles the mean difference was 1.40; at 1000 cycles the mean difference was 0.80, and at 2000 cycles it was 0.20. In this group, the descending series resulted in a threshold measurement either equal to, or 5 db. higher than, the ascending measurement. At 2000 cycles, the descending method gave a lower mean threshold, in general.

I found in a previous study that thresholds for normal hearing persons showed a difference of 2.10 db. between the same two methods of measurement for the 1000 cps tone. It was further discovered that the descending series resulted in a lower threshold measure for every individual. This is directly opposite to the results obtained in the present investigation.

Conclusions

It is apparent that the malingerers utilized in this investigation produced results significantly different from either the normal group or the group with true organic hearing losses in their threshold measurements during this technique. Results showing the descending threshold to be higher than the ascending threshold should be considered suspicious if the difference exceeds 5 db. More specifically, a difference of 10 db. or greater between the two thresholds is highly indicative of nonorganic involvement. On the other hand, differences of 5 db. or less cannot be considered as proof of nonmalingering.

This test has been used with amazing success as a rapid and simple technique to indicate possible nonorganic loss in the clinical situation. It should be emphasized that the use of this test must be limited to its utilization as a screening procedure. It is valuable in detecting nonorganic losses where there may be no reason to doubt the patient's responses. Furthermore, it has no use as a quantitative measure of the degree of nonorganic involvement, although there are indications that the greater the difference between the true organic threshold and the reported nonorganic threshold, the greater will be the difference between the ascending and the descending thresholds. It should not be considered a positive indication of true organic loss where a suspected patient may fall within normal limits in his response to this procedure.

Where a patient shows discrepancies from the expected normal responses, it is recommended that a battery of tests for nonorganic loss be administered to the patient before a positive diagnosis is made.

3614 Fairmount St.

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Case Reports

Combined Internal-External Laryngocele Successfully Treated by a One-Stage Surgical Procedure

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Combined internal-external laryngocele is very rare. Taylor [1] in 1944 stated that in the 66-year period of the Academy meetings only one case was presented.

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The external variety is most prevalent. A review of the literature by O'Keefe [2] for the period 1940-1949 revealed 14 reported cases; 10 were of the external variety and 4 of the internal variety. Over-all total to date is about 75 cases.

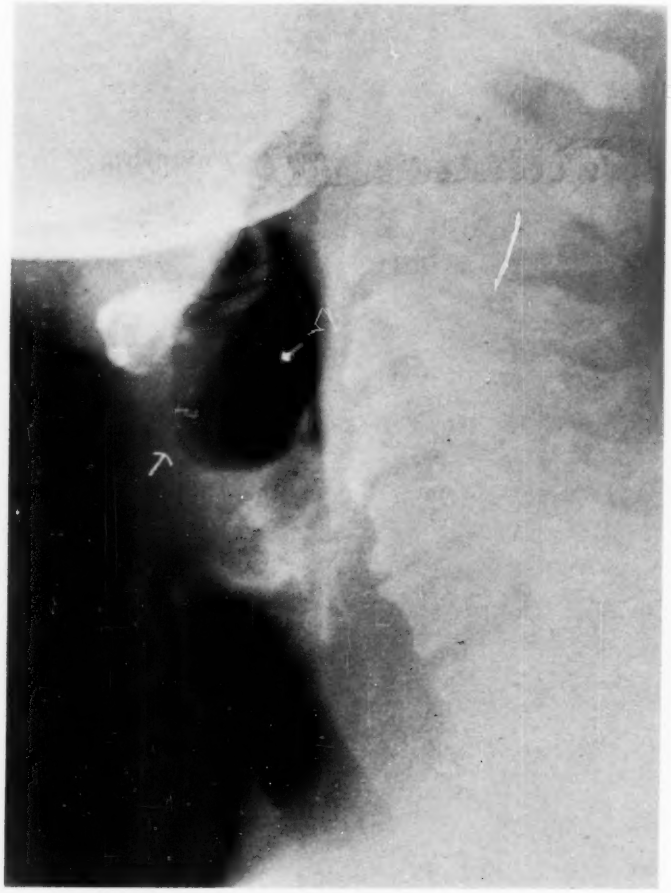

Fig. 1.—Lateral view of larynx showing external laryngocele superimposed over internal laryngocele (indicated by arrows).

In reviewing the literature one encounters numerous surgical techniques in the treatment of the internal and the combined varieties of laryngocele. As for the external variety, the consensus is excision of the sac through a lateral neck incision, the stalk being amputated at the level of the thyrohyoid membrane.

It has been customary to approach the internal variety through either a laryngofissure or a lateral thyrotomy. Most surgeons prefer to remove the posterior third of the thyroid cartilage to get a wider exposure. O'Keefe [3] in 1952 devised a new approach through the thyrohyoid membrane in a case of internal laryngocele in order to avoid splitting the thyroid cartilage and the inevitable tracheostomy.

In the case presented here I have followed this technique. However, my patient had the combined variety of laryngocele, and the good result obtained indicates that this technique is suitable to all cases.

Report of Case

The patient is a 42-year-old lawyer. He first consulted me when I was in Beirut, Lebanon, four years ago. He gave a history of hoarseness, sensation of a mass in the throat, and spells of dyspnea of two years' duration. Examination revealed a sausage-shaped swelling, measuring 3 by 1 cm. on the right side of the neck just below and parallel to the ramus of the mandible. This was tympanitic to percussion. The patient's voice was somewhat hoarse. Indirect mirror laryngoscopy revealed a smooth swelling (3 cm. in diameter) involving the right ventricular band and aryepiglottic fold and hiding the right vocal cord from view.

Fig. 2.—Posteroanterior view of neck showing globular air shadows of external and internal laryngocele.

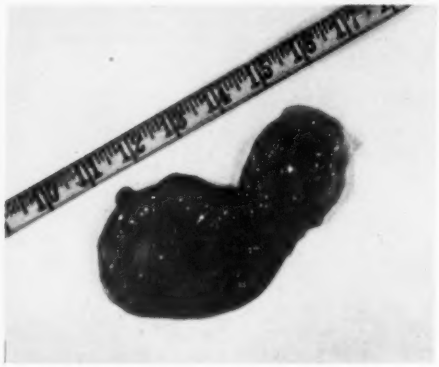

Fig. 3.—Hourglass appearance of combined external-internal laryngocele. Actual size before excision was one and a half times larger.

The patient was asked to cough while the mirror was in place, and the swelling almost doubled in size. By applying pressure and counter-pressure on the side of the neck he was able to reduce the size of the swelling. Seemingly he had mastered this maneuver and utilized it every time he was in respiratory difficulty. The maneuver was accompanied by an audible escape of air.

A clinical diagnosis of laryngocele (internal and external) was made, and x-ray films confirmed the diagnosis.

Surgery was advised but refused. I lost track of the patient until August, 1956, when he wrote and asked me to perform the operation.

The operation was performed at the American University Hospital, Beirut, Lebanon, on Aug. 24, 1956. Every precaution was taken during the anesthesia. I was at hand with the anesthetist. A needle was introduced intravenously, and when we were ready to inject the thiopental (Pentothal), the patient was asked to empty the internal sac, as he knew best. A direct laryngoscopy revealed the sac to have reduced in size, and enough space was present for the introduction of a Magill tube. The thiopental with a curare preparation was then injected, and the patient was intubated without much trouble. NO_2-O_2-ether anesthesia was subsequently used.

An incision 5 cm. in length parallel and about 3 cm. below the ramus of the right mandible was made, extending from the anterior border of the sternomastoid muscle to the midline of the neck. The superficial fascia and the platysma muscle were cut. Dissection was carried down with exposure of the right submaxillary salivary gland. Beneath and below this gland the external air sac came into view. Careful dissection was carried out until the whole external sac was exposed. The part passing through the thyrohyoid membrane was outlined after the ribbon muscles were cut. The superior laryngeal nerve and vessels were exposed and carefully avoided. The thyrohyoid membrane was cut along the upper margin of the thyroid cartilage, and the internal sac came into view. By the aid of peanut sponges one could dissect the sac from the ventricular band without entering the laryngeal lumen. Dissection was completed, and the stalk of the internal sac was followed into the ventricle to its base, held by a mosquito clamp and amputated. The external and internal sac came out in one piece. It was hourglass in shape (5 by 2 cm. in size). The amputated stump was sutured with a purse-string suture and invaginated. The thyrohyoid membrane was then sutured back and the ribbon muscles repaired. A Penrose drain was left in the neck wound. No tracheostomy was necessary. After the Magill tube was removed, direct laryngoscopy was performed. No blood was noted in the larynx and trachea. There was only a laxity of the right ventricular band and no mucosal tears.

The pathologist's report read as follows: "Specimen consists of a cyst pinkish in color and soft in consistency, measuring 5 by 2 by ½ cm. The anterior surface is glistening and smooth. Cut section reveals no gross lesion in the inner surface.

"Histologically the cyst wall is composed of fibrous tissue. The lining is respiratory squamous, pseudo-stratified, columnar or mucin-producing epithelium. Patches of inflammation are seen in the wall."

The patient had an uneventful recovery and was discharged on the seventh day. Mirror laryngoscopy on discharge revealed moderate edema of the right ventricular band and aryepiglottic fold. By the 20th postoperative day this edema had almost completely disappeared. Both vocal cords moved quite freely, and the patient's voice was normal.

Three months ago I received a communication from the patient informing me that he had had no recurrence and had been completely relieved of all his symptoms.

Summary

A case of combined internal-external laryngocele is presented. A one-stage surgical technique first suggested by O'Keefe was successful in the treatment.

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Branchiogenic Carcinoma

M. STUART STRONG, M.D., and SHELDON C. SOMMERS, M.D., Boston

The occurrence of malignant degeneration within the confines of branchial remnants has been the subject of discussion for many years. Formerly, the diagnosis of primary carcinoma of the neck, probably arising in branchial remnants, was not an unusual one. However, in retrospect, it has been found under close scrutiny that in very few of the cases in which the diagnosis of branchiogenic carcinoma had been made can the diagnosis be upheld.

The rarity of this condition has been emphasized by Martin,[1] who has doubted if the diagnosis should ever be made. No tissue in the body is exempt from the possibility of malignant degeneration, and of course squamous epithelium and lymphoid follicles, both of which are found in branchial cysts, are notoriously prone to undergo neoplastic change. However, Martin feels that the only adequate proof of a true branchiogenic carcinoma can be the demonstration of cancer arising in the wall of a branchial cyst. This certainly should be the essential criterion upon which the diagnosis is based.

Report of Cases

CASE 1.—The first patient was a 59-year-old white woman, who gave a history of having had a painless lump in the left side of her neck for about six weeks prior to examination. The patient had no other symptoms referable to the head or neck but she was considerably concerned because the swelling appeared to be gradually increasing in size.

Examination revealed the presence of a smooth, well-defined, elastic swelling measuring about 4 cm. in diameter. The mass was situated anterior to the anterior border of the left sternomastoid muscle, so that the bifurcation of the common carotid artery and the greater cornu of the hyoid bone could not be palpated. The mass had limited mobility but was not actually fixed. No other masses could be palpated in the neck. The remainder of the ear, nose, and throat examination revealed the presence of a very small and inconspicuous 0.5 cm. ulcer situated at the junction of the base of the tongue and the left lateral wall of the pharynx immediately adjacent to the tip of the epiglottis. This ulcer was particularly inconspicuous because the patient had a large amount of lingual lymphoid tissue. Palpation of the ulcer, however, revealed that it was markedly indurated (Fig. 1).

A tentative diagnosis of carcinoma of the base of the tongue with cervical metastasis was made. A biopsy specimen was taken from the ulcer and was found to be epidermoid carcinoma, Grade II, in association with a large number of lymphatic cells—the so-called "lymphoepithelioma" (Fig. 2).

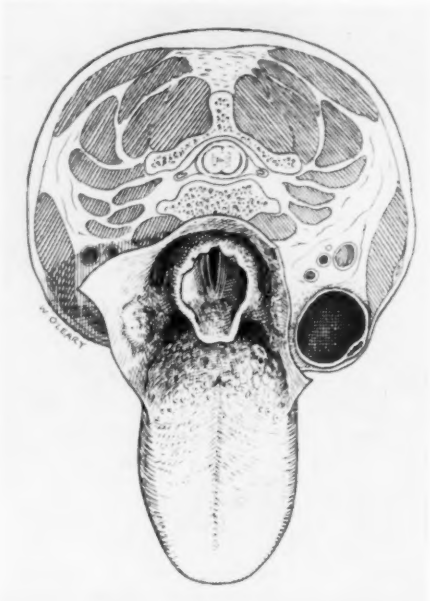

Fig. 1.—Diagram showing the relationship of a very inconspicuous malignant ulcer at the base of tongue and the associated cervical mass.

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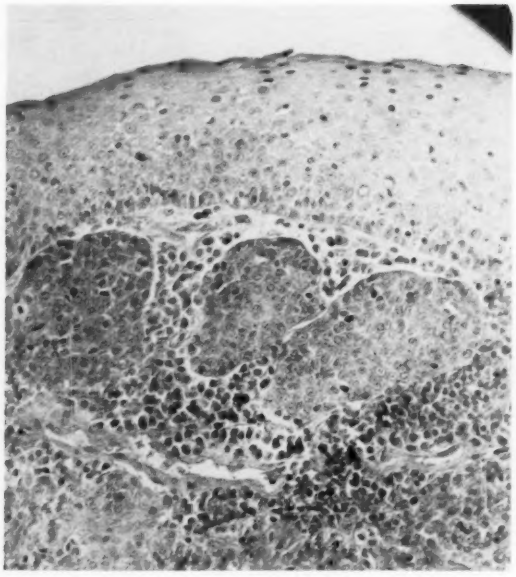

Fig. 2.—Nests of epidermoid carcinoma cells surrounded by numerous lymphocytes beneath the normal squamous epithelium of the base of the tongue. Hematoxylin and eosin; × 200.

Because of a general feeling that a primary epidermoid carcinoma occurring in the immediate proximity of lymphoid tissue carries a poor prognosis even when the most radical surgical maneuvers are used, it was decided that external radiation should be relied upon and surgery withheld unless the cervical mass failed to show prompt evidence of response to radiation.

Prior to commencing x-ray therapy, a prophylactic tracheotomy was performed so that the radiotherapist could continue his treatment without concern for the laryngeal airway. The radiation was given through three external portals which were concentrated upon the primary lesion, and after a calculated tumor dose of 3000 r had been given it was noted that the primary ulcer had been completely obscured from view by edema and radiation reaction. However, the cervical mass which was included in the left lateral portal did not show any signs of regression and, in fact, appeared to be slowly increasing in size. This was a most unexpected course of events because it was felt that the tumor would almost certainly be radiosensitive.

In the absence of any other palpable cervical nodes, it was decided to do a biopsy excision of the cervical mass. At operation as soon as the investing layer of the deep cervical fascia had been incised, a pale, smooth, well-encapsulated tumor was found, anterior to the anterior border of the sternomastoid muscle. It was easily separated from the internal jugular vein and carotid artery. The base of the mass was centered over a point below and behind the greater cornu of the hyoid bone and anterior to the bifurcation of the carotid. No distinct pedicle was attached to the mass. The mass was removed in toto; a search was made for other enlarged cervical lymph nodes but none were found.

The 4 cm. mass was tense and elastic. When the mass was opened, a large quantity of opalescent, straw-colored fluid escaped, leaving only a very thin-walled and collapsed cyst. From this it was concluded that the lesion was in fact a cervical lymph node which had become involved by metastasis from the primary carcinoma at the base of the tongue and that the tumor had been broken down by radiation.

Pathologic study, however, revealed that the lesion was not a lymph node but, in fact, a branchial cyst, the wall of which presented many areas of fibrosis and foreign-body giant-cell reaction to cholesterol. The lining membrane was a thin layer of squamous cells. The cyst wall was thin and smooth throughout. A close inspection of the cellular components of the cyst wall, however, revealed several nests of epidermoid carcinoma. These malignant cells appeared to have no connection with either the outside or the inside of the cyst wall. Because of this, a diagnosis of epidermoid carcinoma arising in the wall of a branchial cyst was made. Thus, in addition to the primary lymphoepithelioma of the base of the tongue, the patient also had a primary branchiogenic carcinoma (Fig. 3).

The course of external radiation was continued until a calculated tumor dose of 8400 r had been given.

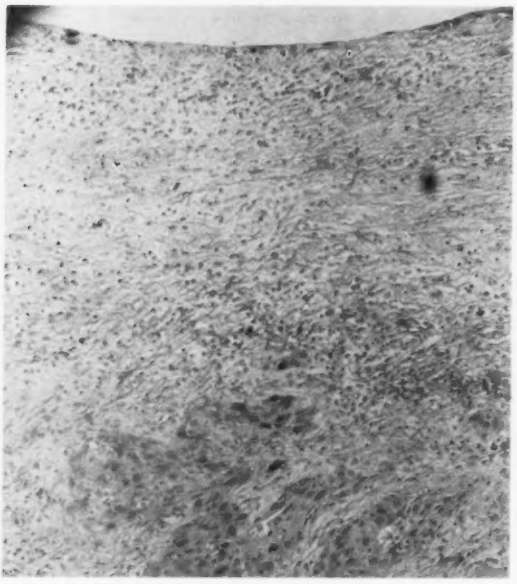

Fig. 3.—Wall of branchial cyst showing a single layer of lining cells, lymphocytic infiltration, and several areas of epidermoid carcinoma; × 30.

CASE 2.—The second case was a 66-year-old man who complained of a lump in the right side of his neck for five weeks. The mass was situated deep to the anterior border of the sternomastoid muscle at about its midpoint. No other abnormal findings were encountered in the ear, nose, or throat.

The mass was excised, and the wound healed uneventfully. Gross examination revealed that the mass was a thin-walled cyst measuring 3×2×2 cm.; it was grayish pink in color and filled with grayish fluid in which floated flecks of white matter.

Pathologic study revealed a thin-walled cyst lined by squamous epithelium; the wall was composed mostly of fibrous tissue and contained several areas of lymphoid infiltration. The cyst was clearly seen to be of branchial origin. While most of the epithelium was entirely normal, at one point it was seen to have undergone malignant change and to

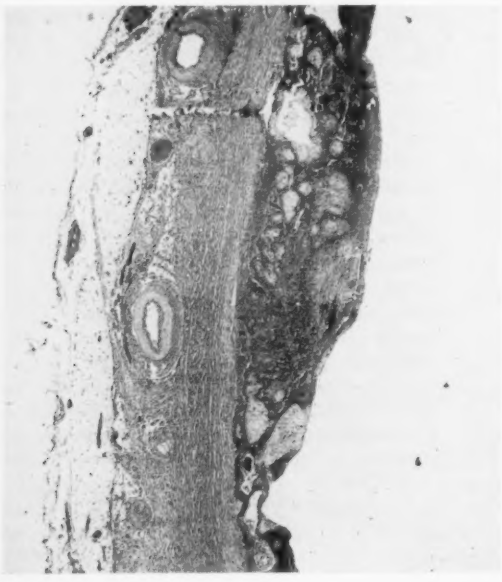

Fig. 4.—Fibrous wall of branchial cyst showing, under low-power magnification, a relatively large amount of piled-up malignant epithelium; × 20.

Fig. 5.—Medium scanning power, view of cyst wall showing the dense lymphocytic infiltration and an island of malignant cells still partly attached to the uninvolved squamous lining; × 30.

have infiltrated the wall of the cyst. An unequivocal diagnosis of early epidermoid carcinoma arising in a branchial cleft cyst was made (Figs. 4, 5, 6, and 7).

The patient was followed for two years, during which time he did well until he developed hoarseness and was found to have an epidermoid carcinoma of the larynx. This was treated in another hospital by external radiation, which did not control the cancer, and he died, one year later.

Comment

These reports are of interest because it is almost axiomatic that an unexplained mass in the neck represents a metastasis

Fig. 6.—Higher-power view of epidermoid carcinoma cells "budding off" from the squamous epithelium into a large collection of lymphocytes; × 80.

Fig. 7.—Higher-power view elsewhere showing malignant epidermoid cells beneath an intact cyst lining of squamous cells; × 60.

from a primary carcinoma in the upper respiratory tract until proved otherwise.

There can be no doubt that primary carcinoma arising in a branchiogenic cyst does exist as a clinical and pathological entity. Willis [2] in his "Pathology of Tumors," reports that on only two occasions has he made the diagnosis of branchiogenic carcinoma. He feels that this is an extremely rare entity. Similarly, Eggston and Wolff [3] in their "Histopathology of the Ear, Nose and Throat," recall several cases of indisputable epidermoid carcinoma of the branchial cyst wall, and on two occasions have found lymphosarcomatous degeneration of the cyst wall.

Lahey [4] felt that because of the epithelial elements in a branchial cyst there was always the danger of malignant degeneration present. He, however, agreed that this danger was low.

In 1952, Hill and Goodof [5] reported an interesting case in which a branchial cyst was removed and on two occasions recurred, requiring second and third operation; it was found that an epidermoid carcinoma, Grade II, had developed within the wall of the recurrent cyst. Of particular interest is the fact that one year later the patient developed a carcinoma of the tonsil on the same side. This case, therefore, also presented the feature of multiple malignant neoplasia occurring in the pharynx, following a carcinoma in the branchial cyst one year previously.

It should be recalled that branchial cysts arise usually from the second and sometime the first pharyngeal pouch. In this way a branchial cyst is composed essentially of the same tissue elements as the wall of the pharynx or larynx which arises from the same or adjacent pharyngeal pouch. This identical embryologic inheritance of a branchial cyst and the walls of the pharynx or larynx would then make them equally susceptible to any intrinsic or systemic carcinogenic factor.

Warren and Ehrenreich [6] have calculated from a detailed statistical study that a susceptibility or predisposition to carcinoma exists in certain persons. They found that there was a greater tendency for multiple malignant lesions to occur in the same system than in dissimilar systems. The cases of multiple malignancies occurred more fre-

quently than the expected incidence based on chance alone.

Prior to this report of Warren and Ehrenreich, Lund [7] in 1933 had found in a long series of carcinoma of the mouth that the occurrence of a second carcinoma was 15 times as common as it should be if chance alone was the only factor.

These two cases reported, then, represent a simultaneous occurrence of multiple malignant tumors occurring, on the one hand, on the base of the tongue or in the larynx and, on the other hand, in the embryologic extension of these organs, namely, a branchial cyst.

Summary and Conclusion

It should be emphasized that the finding of a cervical swelling of undetermined origin calls for a very thorough inspection and examination of the entire upper respiratory tract and a search for a possible primary neoplasm which might have metastasized to the neck. The diagnosis of branchiogenic carcinoma should not be made except in cases in which there is both gross and microscopic evidence of a true branchial cyst and carcinoma is found within the cyst wall.

The occurrence of a branchiogenic carcinoma in association with a carcinoma at the base of the tongue and in another case with a laryngeal carcinoma are as unusual as they are interesting. It is explicable on the grounds of the known propensity of mucosa arising from one or more of the pharyngeal pouches to develop multiple cancers.

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Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Stanton A. Friedberg, M.D., President

Monthly Meeting, Feb. 3, 1958.

Laryngocele: Report of a Case. ROBERT BORKENHAGEN, M.D. (by invitation), and MAURICE F. SNITMAN, M.D.

A laryngocele is a hernia of the larynx; it arises from an appendix, or saccule, of the laryngeal ventricle. The herniation of mucous membrane may be toward the larynx, away from the larynx (laterally into the neck, that is), or it may be both, into the larynx and out to the neck.

This appendix in man contains mucus-secreting glands, and ordinarily has no other function. It is a three-dimensional structure and is better understood if its boundaries are visualized: "The saccule (appendix) is bounded laterally at its base by fibers of the thyro-arytenoid muscle and by the connective tissue of the ary-epiglottic fold. Medially it is shut off by the mucous membrane and some of the connective tissue of the ary-epiglottic fold. Superiorly, anteriorly and posteriorly the limiting walls are provided by connective tissue of the same fold, while inferiorly there is free communication with the ventricle" (Negus).

An etiologic factor in development of a laryngocele may be a congenitally long appendix, which predisposes to herniation. An exciting factor may be related to excessive laryngeal air pressures developed in persons using the voice excessively. The original description, in fact, of laryngocele was by a French surgeon, Larrey, who in 1829 noted the condition in persons employed to chant verses of the Koran.

In a comparative sense, the appendix of the ventricle is not well developed in man. In animals, however, it may become quite large. In some apes, for instance, it extends as far as the axilla under the pectoral muscles and is used as a compensatory respiratory mechanism.

Laryngocele is believed to be commoner in women and children, although abnormal use of the voice is commoner in occupations of men. The condition may be unilateral or bilateral. The gross pathology is an air-filled or fluid-filled sac which may communicate with the ventricle. Microscopically the tissue is made up of pseudostratified ciliated columnar epithelium. This is important in differential diagnosis where dermoid, branchial, or other cystic structures are found in the neck. In an only reported case, carcinoma was found in association with laryngocele.

Treatment is surgical and is motivated because of the possibility of respiratory obstruction, pressure phenomena in the neck, or infection of the laryngocele.

A case of combined external and internal unilateral laryngocele in a 37-year-old white man is presented. The patient had noted hoarseness and swelling in the left neck for about two months. The diagnosis was made from the history, roentgen findings, and laryngoscopic observation.

Treatment was surgical, using an external approach. A "leaf" technique was employed. Instead of dissecting the mass as a closed cyst, it was purposely opened, and the edges created a leaf of tissue. This leaf was pursued from its inner or mucosal surface. The base was thereby easily elevated into view. Ligation and excision could more easily be performed under direct view. The laryngeal mucosa was not opened in the dissection. A tracheotomy was not necessary.

Discussion

DR. BURTON SOBOROFF: It is most interesting to me that laryngoceles in almost all of the cases show identically the same picture. As a matter of fact, as reported in the *Annals*, Lyman Richards reported several cases, and others have reported these cases. If you see their x-rays, they have almost an identical picture. You could superimpose one x-ray on another.

One of the things which is particularly interesting is the barium swallow, since the barium appears inside the sac which supposedly originated within the ventricle. We noted on the barium swallow that it appears inside this sac, and this may be different because the sac arises from the saccule of the ventricle. In the youngster this arises a little higher than in the adult, at about the level of the hyoid. Either there is bulging internally, or it enlarges outward into the thyrohyoid membrane, producing the bulging in the neck, or there may be a combined type.

There was one with external bulging, and we thought we had a laryngeal fissure, but it proved to be only a bulging, a swelling of the false cord

producing obstruction and marked hoarseness. There was no swelling of the neck at all. Some of them can be primary, as this one appeared. Some can be secondary to an infection, a pyocele, or a laryngocele. Some are dissected directly, and when you open the leaf you can pass a probe. The surgical treatment is that of a hernia—that is, a high ligation of the sac, or, in cases where there is bulging, a low ligation. (Slides were shown).

[Slide]: This man, who was operated on at the University, had bilateral involvement and thickening of the vocal cords, and for a long period of time no specific diagnosis was available. The findings were of some swelling in the neck and a laryngocele. Here you see the laryngocele, the vocal cords, and the bulging in the neck. We did a transverse incision.

[Slide]: This is the right side of the neck with the sac appearing here. Usually it includes the thyrohyoid muscle.

[Slide]: This shows the bulging of the sac on the left. In this case there appeared to be a bulging of the pharynx. Finally we opened it and grasped it and dissected it down to the pharyngeal mucosa lying beneath it. The neck is opened, and the thyroid cartilage laid over, thus exposing the larynx.

[Slide]: The sac is then opened, and a probe can be passed into the pharyngeal ventricle.

[Slide]: This is the sac dissected away, just below the hyoid. As you know, it is easier and more thorough in dissection to open the sac into two leaves and follow the dissection downward. We use high ligation, just as high as possible, and eliminate the entire hernia.

[Slide]: Finally, these are the two specimens, one from each side, which showed pseudostratified ciliated columnar epithelium, the same as in the larynx, the ventricle, and the pharynx.

DR. PAUL H. HOLINGER: I want to compliment Dr. Borkenhagen for his presentation and his beautiful photographs, and Dr. Snitman, for his description of the surgical technique.

I should like to ask a question in regard to diagnosis. Are the air-containing sacs true laryngoceles, or might they not be pharyngoceles? In a recently reported large series they were called pharyngoceles rather than laryngoceles.

Another point is in regard to therapy. In general, the best advice is that the sacs should be removed, and usually there is no substitute for the external surgical approach. However, I might mention that on two occasions we have aspirated large laryngoceles found on endoscopic examination. They contained a large amount of mucus, and disappeared completely on aspiration with a large-bore needle. Both patients were more than 75 years of age and were hoarse and dyspneic, and this simple endoscopic procedure completely alleviated the symptoms.

DR. JOHN R. LINDSAY: The essayists have not said much about the cause of the laryngocele. It is significant that this appendix comes off between the true and the false cord. Glottic closure during greatly increased intrathoracic pressure is maintained chiefly by approximation of the false cords, since they have some valvular action against the escape of air from the chest. In the case which I reported some years ago, the laryngocele would dilate on muscular activity of the upper extremity. In the ape, the upper extremity is extremely well developed. The activity of this upper extremity, necessitating fixation of the chest by glottic closure, probably contributes to the development of the laryngocele. In people who chanted the Koran from high places, the false cords were probably used in this particular method of producing the voice, thus exposing the laryngeal ventricles to the increased intrathoracic pressure and predisposing to a laryngocele.

DR. BURTON SOBOROFF: Just a word about pharyngocele and laryngocele. I believe that these may well be pharyngoceles, but in addition this man demonstrated this trouble in the false cord. He was obstructed and had swelling of the false cord which was large and very severe. The sac led to the ventricle in between the cords, and both may have been present.

The First and Second Branchial Arches: Their Developmental History and Adult Contribution to the Auditory Apparatus in Man. BARRY J. ANSON, M.D. and THEODORE H. BAST, M.D.

The manner in which the branchial skeleton of the aquatic and amphibious creatures is salvaged and remodeled to meet the requirements of vertebrates far higher in the phylogenetic scale has been for many years a subject of laboratory study and resultant debate.

In the course of their investigation, the present authors have dealt with this problem as it concerns the developmental sources of elements which become part of, or closely related to, the human otic capsule and temporal bone. This means, of course, that the embryonic and fetal derivatives of the first and second branchial arches have been traced from their genesis in precartilage to the stage in which they assume adult position and form.

Toward the close of the sixth week of intrauterine life, precartilage makes its appearance in the region of the first and second branchial arches. Prior to this stage the formative mesenchyma is concentrated in predictive masses with indefinite histological limits. Differentiation thereupon proceeds rapidly, true cartilage being formed during the seventh and eighth weeks.

In the fetus of 10 weeks the primordial masses have assumed recognizable form. During this period, changes in Meckel's cartilage precede those in Reichert's cartilage. Certain related structures have already been precociously formed in bone—the mandible, the anterior process of the malleus, and the tympanic ring (attaining osseus structure in the seventh, eighth and ninth weeks, respectively).

Meckel's cartilage may be subdivided into three main areas in which different types of metamorphosis occur: The dorsal end gives rise to the malleus and incus, through endochondral ossification; the middle portion becomes the anterior ligament of the malleus, the sphenomandibular ligament, and a fibrous adjunct to the periosteum of the body of the mandible; the ventral end, encroached upon by the adjacent bone of the mandibular tip, is transformed into bone, the latter then being incorporated into the anterior part of the osseous mandible.

Soon after the ossicles have been fully formed in bone, separation of the malleus from the middle segment of Meckel's cartilage begins, by a process of deorganization which is followed by one of conversion of this tissue into the suspensory ligament of the malleus and the sphenomandibular ligament. Segregation of the differentiating tissue of the ligaments from a primordial tissue goes on currently with fusion of the anterior process with the neck of the malleus. The remainder of the middle part of Meckel's cartilage serves as an adjunct to the periosteum of the mandible; the distal part contributes to the bone at and near the symphysis.

The mandible begins to ossify during the sixth week (at a time when the branchial arches are just recognizable in precartilage). Membrane bone appears near the future mental foramen and at an appreciable distance from the anterolateral aspect of Meckel's cartilage. With further osteogenesis a lamella of bone is formed along the outer alveolar wall. An inner ridge of bone, continuing posteriorly, terminates as the lingula of the mandible. In the ninth week the membranous mandible acquires an embryonic tissue of an unsual type on the posterolateral aspect of the condyle. This tissue passes into membrane bone in the lower part of its extent in the mandibular ramus; the midportion calcifies without enlargement of excavation by osteogenic buds, then to be removed by osteoclasts and replaced by membrane bone; in the condylar portion it differentiates into cartilage and then proceeds through changes characteristic of enchondral ossification. Similar tissue occurs in the coronoid process, at the symphysis, and along the alveolar margins.

Like the derivatives of Meckel's cartilage, those of Reichert's bar are varied: cranial, in transitorily forming part of the facial canal, and in giving rise to the styloid process; ossicular; ligamentous, in providing the tissue for the stylohyoid ligament.

Reichert's bar remains cartilaginous through almost the entire fetal period (into the 34th week); in the fetus at term it is ossified only in the proximal segment, where it is continuous cranially with the otic capsule. Even in some adults, cartilage persists in the stylohyoid ligament near the tip of the styloid process of the temporal bone.

While the canalicular division of the otic capsule is still cartilaginous, in the fetus of 21 weeks, the future canal for the facial nerve is merely a shallow furrow. The tissue in which the nerve and stapedial muscle are lodged is highly vascularized, and will remain so. In a fetal specimen 13 weeks older, the deepened sulcus has acquired a lateral wall, thus becoming a true canal. In this process, the canal becomes separated from Reichert's cartilage, which in earlier stages constituted an outer boundary.

Discussion

Dr. Theodore H. Bast: There is really very little that I could add to the presentation. Dr. Anson has a very pleasing way of presenting the material.

This problem has been of interest to us partly from the purely scientific standpoint, and, of course, also from the standpoint of clinical procedures at the present time. But from a purely scientific point of view, we have always heard that the stapes is produced from the second visceral arch, and the malleus and incus from the first visceral arch. There is no question from our observations that the malleus is a distinct continuation of Meckel's cartilage or the first arch, and, of course, we have all this time felt that the incus possibly also belongs to this first arch. Dr. Anson also mentioned that the stapes has possibly arisen from two sources: the basilar part, or footplate, from the otic capsule, and the crura from the second visceral arch. That has been the conventional way of putting it. We have been very much interested in the human to see if we could trace any continuity between Meckel's or Reichert's cartilages and the formation of these ossicles. So far that story is not complete. We still have more work to do, but at least we have demonstrated that Reichert's cartilage shows no continuity with the stapes whatsoever, there is a little mass of cellular material between the stapes and the cartilage known as the interhyal area, and in some of the lower forms that apparently goes over to cartilage, but it does not in the human as far as we can determine. It is transformed into the stapedius muscle. Thus, while the stapes is possibly a product of the second arch, and that can hardly be questioned, it is certainly not a direct product of Reichert's cartilage.

The cellular part can form the stapedius muscle. However, there is another problem about which I can say nothing positive at the present time. The incus seems to be more intimately associated with the stapes, from what I can see in the human embryo, than it is to the malleus. The stapedius muscle in many of the cases has one strip of attachment to the stapes and the other strip to the incus. If this muscle with its two tendons is derived from the interhyal mass, the incus should be derived from the second arch, the same as the crura of the stapes. Certainly the second arch is innervated by the seventh nerve, and, since the seventh innervates the stapes muscle, it would almost seem that the incus should be of second arch origin. Furthermore, the chorda tympani nerve, a branch of the seventh cranial nerve, passes between the incus and malleus. We cannot answer that at the present time. We hope that in some of the earlier embryos we may be able to get further information.

DR. ROBERT HENNER: I should like to make a point which this discussion has stimulated: In three recent cases of what we call congenital cholesteatoma, without adequate information of the inflammatory nature of the origin of the cholesteatoma seemed to be around the head of the stapes, right at the differentiation from Reichert's cartilage. In the latter site, according to your lecture, it could easily be a pinched-off epithelial inclusion, and in this way could be the common site of a so-called congenital cholesteatoma.

The other matter that came to my mind is that if the incus is possibly part of Reichert's cartilage, and in almost every congenital stenosis and atresia we find it involves both the incus and the malleus, therefore, it would seem that they would have a common origin. I wonder if this was taken into consideration in your study of the embryonic material.

DR. BARRY J. ANSON: In reply to the first comment, there is no reason to doubt the common statement that embryonic masses may be the source of pathological tissues in the adult. At present, this is a matter of surmise, since we do not possess knowledge of the intermediate steps in morphogenesis. A similar statement would be applicable in reply to the second comment.

In reply to the third comment, so much information has been derived from recent restudy of the developmental and adult anatomy (normal and pathological) of the ear and temporal bone that I am forced to admit that standard textbook accounts of otological anatomy are obsolete.

Photography of the Nasopharynx. (Slides) DAVID F. AUSTIN, M.D. (by invitation).

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Monthly Meeting, March 3, 1958.

Paralysis of the Larynx. LOUIS H. CLERF, M.D.

The cause of paralysis of the larynx rarely is found within the larynx itself. It should be looked for within the cranial cavity, the neck, or the thoracic cage. It might, therefore, be considered as a symptom or local manifestation of disease elsewhere.

The views expressed by Onodi are generally accepted, namely, that all the muscles of the larynx except the cricothyroid are innervated unilaterally by the corresponding recurrent laryngeal nerve. The cricothyroid is supplied by the corresponding external or motor branch of the superior laryngeal nerve. According to Todd and Vogel, the internal branch of the superior laryngeal division contains motor fibers which supply the arytenoideus muscle.

Terminology regarding the position of a paralyzed cord appears complicated, and references are made to many positions. Obviously there are three positions that a vocal cord can assume, namely, in the midline or adduction, in a lateral position or abduction, and in a position between abduction and adduction. Usually there is some relaxation of the paralyzed cord, but this may be so slight that there is no interference with the speaking voice. One also must take into account the bilateral innervation of the arytenoideus muscle which may produce tilting of the paralyzed arytenoid toward the midline, suggesting that there is adductor movement. In addition, consideration must be given the cricothyroid muscle, which, although primarily a tensor of the vocal cord, also exerts some adductor action.

Motor paralyses commonly are classified as central or peripheral in origin. About 10% are central and are caused by progressive bulbar paralysis, tabes, syphilitic arteritis, disseminated sclerosis, acute anterior poliomyelitis, syringomyelia, vascular accidents, trauma involving the base of the skull, tumors, and abscesses. Since the origin of the vagus in the floor of the fourth ventricle is in close relation to the centers of the glossopharyngeal and spinal accessory nerves, paralysis of the larynx of central origin commonly is associated with paralysis of other groups of muscles.

In peripheral paralysis the vagus nerve may be involved at the jugular foramen, the superior laryngeal branch may be involved in the neck, and

the recurrent or inferior branch in the neck or mediastinum.

A diagnosis of paralysis usually can be made without difficulty by mirror rather than by direct laryngoscopy. Observing the cords during inspiration and phonation will give one definite information. If there is question, the cords should be observed during cough. This is of great value in the diagnosis of so-called functional paralysis.

Determination of the cause of the paralysis is more difficult. A complete history, physical examination, neurologic examination, roentgen studies of the neck and chest, and serologic studies of the blood may be required. Often a functional study of the swallowing function is indicated. Patients with paralysis of the larynx frequently have dysphagia due to interference with the protective function of the larynx. Direct laryngoscopy, bronchoscopy, and esophagoscopy may be required. In spite of complete studies, a certain number of cases, approximately 8% to 12%, will remain unsolved etiologically.

The fate of the paralyzed cord is dependent on the cause. In paralysis following operation or injury or occurring as a result of an acute infection or toxic cause, there may be restoration of function in from four to six months. One rarely sees function return after six months. A guarded prognosis should, therefore, be given, and surgical treatment, except tracheotomy, probably should be deferred until at least six months have elapsed.

Treatment presents a difficult problem. The cause usually cannot be removed. There is no known medical treatment that will restore function. Relief of symptoms often may be secured by surgical measures.

Discussion

Dr. Paul H. Holinger: I have two questions. First, I should like to thank Dr. Clerf for his discussion of this subject. The first question is, do you have any tricks in the diagnosis of paralysis in infants, where the laryngoscope is likely to cause fixation of the cord? Second, what is your modification of the King operation which you mentioned several times without describing?

Dr. Louis H. Clerf: Diagnosis of paralysis of the larynx in the infant is difficult, since mirror laryngoscopy is not possible and direct laryngoscopy is the only available means of examination. If the epiglottis is not of a markedly overhanging type, placing the tip of the spatula at the base of the tongue may give sufficient elevation of the epiglottis so that the cords can be seen. If that is not possible, one may employ a short full lumen esophagoscope passed behind the epiglottis and then rotated so that the lip is posterior. This often is helpful.

The modified King operation refers to fixation of the arytenoid cartilage to the posterior border of the thyroid cartilage. King originally used the omohyoid muscle, which was secured to the arytenoid. In one of my early cases I could not find the omohyoid muscle, which apparently had been cut at the time of the thyroidectomy. I, therefore, sutured the arytenoid to the soft tissues along the posterior border of the thyroid cartilage. This was the best result I had secured up to that time. In all subsequent cases I drilled a hole near the posterior border of the thyroid ala with a dental burr and secured the arytenoid with a black silk suture.

The Larynx and Voice: Laryngeal Physiology Under Daily Stress. (Motion Picture). Hans Von Leden, M.D. and Paul Moore, Ph.D.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Monthly Meeting, April 7, 1958.

Fundamentals of the Radiologic Examination of the Temporal Bone. Wesley E. Compere, Jr., M.D. (by invitation).

Numerous positions have been advocated for the radiologic examination of the temporal bones. Most of these were devised in the preantibiotic era, when acute mastoiditis was the predominant pathology encountered. Although the conventional positions now in use are valuable for the examination of acute mastoiditis, the attic-aditus-antral area (the key area) is so obscured by the dense housing of the labyrinth that they are of little help in the attempt to visualize this area in cases of chronic mastoiditis.

In the Law position, the tympanic cavity is represented by an area of diminished density produced by the superimposition of the external auditory canal on the one side upon the vestibule and internal auditory canal on the other. While topographic views of the antrum can be obtained in such positions as the submentovertex, and the Chamberlain-Towne, the increased antrum-to-film distance produces loss of detail and clarity of projection.

To aid the otologist in more readily choosing between the radical and modified radical procedures, as well as between the various types of tympanoplasty, it is desirable that the relatively early inflammatory, granulomatous, and resorptive fac-

tors be recognized prior to the incidence of the grossly destructive resorption and hearing loss accompanying cholesteatomatosis. In no projection, other than those of Mayer and Owen can the fine detail of the attic-aditus-antral area be obtained. By the addition of the Stenvers position to the Mayer and Owen, every minute portion of the "key area" can be investigated in detail.

These roentgenograms are not so difficult to interpret as to produce in a laboratory committed to a large volume of work. Measurement scales, head-holders, protractors, plumb-bobs, and gadgets in general, are not conducive to readily reproduced films of replicative accuracy. Although these films can be taken on a conventional table, the results are difficult or impossible to reduplicate accurately. By the use of a properly constructed head unit, the patient sits erect in a comfortable position. The apparatus is adjusted to the patient, not the reverse.

The GE Reflex Cassette Tunnel may be readily adapted to this type of work. The tunnel is just large enough to contain the smallest cassette that can hold an 8×10 inch film, plus the extra width necessary to permit a one-half lateral shift of the cassette. The central aperture, through which the film is exposed, is cut through a lead plate and the film may be exposed at its center, or the right and left halves at will. The positioning is done by the mirror and cross method, the central beam being focused on the cross. The area to be studied may be accurately positioned in relation to the cross.

In 1948, Gianturco submitted an appliance which operates with comparative ease in all the desired mastoid positions, with the exception that stereoscopy cannot be attained. This device is not expensive and can be attached to any conventional unit.

The Franklin head unit may be adapted for use in the Mayer and other temporal bone positions. Stereoscopy is easily attained. This is a very convenient, though expensive, appliance.

In 1904, Henle described a position taken laterally from a 15 degree elevation. In this position, or in the more commonly used Law position, taken laterally from an angle of 15 degrees cephalocaudad with 15 degrees dorsoventral rotation, the following landmarks are clearly shown: the root of the zygoma; condyle of the mandible; temporomandibular joint; external auditory canal superimposed on the tympanic cavity, vestibule of the labyrinth, and the internal auditory canal; the cellular structure of the mastoid process; and the sinus plate. The tegmen is well shown, with a small amount of pneumatization between it and the dense bone of the eminentia.

Schuller's position, described in 1905, is taken laterally from a 30 degree elevation. The change in the angulation of the central ray depresses the eminentia to a considerable degree, thus exposing the area of the antrum and epitympanum. The remaining landmarks of the Law position are still seen.

By increasing the cephalocaudad angle to 45 degrees and rotating the head ventrodorsally 30 to 45 degrees (depending upon the shape of the skull), as in the Mayer position, the "key area" is freed from the dense shadow of the labyrinth. The malleus and incus are well seen in the upper tympanic and epitympanic cavities. The cellular pattern of the attic walls is well indicated. The antrum is visible as a small area partially obscured by the eminentia arcuata. The longitudinal line of the crest of the petrosa is now practically vertical.

At operation, the otologic surgeon starts with a definite anatomic mental picture of the anatomy which will confront him. Although the Mayer position projects all the important landmarks, the obliquity of the projection produces such distortion that the surgeon may find difficulty orienting himself. In the Owen position, the projection of the anatomy of the "key area" will most closely meet the surgeon's memory pattern. All the anatomic landmarks of the Mayer are retained; however, the malleus and incus are in a more natural position in the tympanic cavity, the short arm of the incus extending into the aditus. The lateral wall of the attic is visible with a definite pneumatic pattern, and the area of the antrum is visible in a more normal relationship above the eminentia.

The Owen position is taken from a cephalocaudad angle of 25 to 30 degrees, with a ventrodorsal rotation of 30 to 40 degrees, depending upon the cranial type. The Mayer and Owen positions are complementary. The surgeon prefers the Owen, but the roentgen pathologist prefers the combination. We consider the Owen not a devaluation of the Mayer, but an amplification of our roentgen armamentarium.

Whereas the tympanic cavity, attic, and aditus are best visualized in the Owen and Mayer positions, the area of the antrum is seen to the best advantage in the Stenvers position. This position is taken at almost right angles to the Owen, the petrosa being parallel to the central ray in the Owen, but parallel to the film in the Stenvers. The Stenvers projection is taken from a caudocephalad angle of 12 degrees, a very unusual angle in our studies of the temporal bone.

Stenvers position reveals the following anatomic landmarks: the cellular structure of the petrosa, internal auditory canal, cochlea, horizontal and vertical semicircular canals, vestibule of the labyrinth, area of the antrum, dural plate, tympanic cavity, ossicles, condyle, and the mastoid air cells lateral to the antrum and in the mastoid tip.

The best temporal bone films are the result of many factors. For the best detail, one should use

"fine focus" tubes, the smallest possible cones, the Potter-Bucky diaphragm or Lysholm grid, clean screens, fresh solutions, and time-temperature development. The closest possible mastoid to cassette apposition must be maintained at all times.

The most effective method of immobilizing the head is that of seating the patient comfortably. If the posture of the patient is at all strained, the position will be difficult to maintain, no matter what type of headholder is used.

Stereoscopy is particularly valuable for the otologic surgeon who is used to thinking of the temporal bone in three dimensions. The surgeon's familiarity with the radiologic anatomy of the temporal bone will be greatly facilitated if he becomes accustomed to viewing stereoscopic films taken according to a routine, standardized technique.

Summary.—The radiologic examination of the temporal bone is one of the most interesting studies in the field of otology.

By the use of properly constructed cassette holders and properly chosen positions, every portion of the temporal bone can be explored in detail. A brief review of the most useful positions has been presented, and reference has been made to head units suitable for or adaptable to this kind of work.

Representative films demonstrating the pathology in certain common and interesting diseases of the temporal bone have been presented.

Deafness Following Short-Term Exposure to Industrial Noise. SHERMAN L. SHAPIRO, M.D.

The author classifies the existing literature on acoustic trauma into two parts: that dealing with explosion deafness, and that dealing with the effect of chronic exposure to industrial noise upon the ear. He introduces a third group, deafness following short-term exposure to industrial noise, and presents six case histories illustrating this condition. These involved relatively young workmen, four being in their thirties, who had been exposed for periods varying from seconds to eight and a half days to noise from hammer blows; two of the tools were sledge hammers, the others were of the pneumatic variety. In nearly all cases there was a history of working in a confined space. Audiograms and tuning fork tests indicated the hearing loss to be either largely or wholly unilateral to the point of deafness in one ear.

It is the author's belief that special sensitivity to noise was an important factor in these cases. Illustrative records of these cases, as well as of three instances of chronic exposure to noise which were used for comparison, are included.

Discussion

DR. MEYER S. FOX (discussion read by DR. M. M. HIPSKIND): Dr. Shapiro has used the term "acoustic trauma" to describe the following (1) the sudden hearing impairment resulting from intense blasts, explosions and gunfire; (2) the gradual hearing impairment resulting from chronic exposure to industrial noise, and (3) the deafness following short-term exposure to industrial noise. One could add a fourth category to include hearing loss and injury to the auditory apparatus resulting from direct blows to the head and ears as described by Dr. Schuknecht. I feel that the term "acoustic trauma" is particularly confusing and can be interpreted differently in the medical and the legal sense. Therefore, in order to logically distinguish between the various conditions, acoustic trauma should be reserved for the immediate hearing injury produced by one or a few exposures to very intense sounds as blasts or explosions and noise-induced hearing loss should be restricted to describe the slowly progressive, cumulative loss of hearing —always of the nerve type—that develops over a period of months or years of employment and results from "noise exposure." From experience we have learned that "noise exposure" has four important components, all of which contribute to the production of hearing loss. These four components are (1) the over-all sound pressure level reading; (2) the composition or frequency spectrum of the noise; (3) the duration and distribution of exposure during a typical work day, whether the sound is a steady or intermittent impact type, and how often it occurs during the day; (4) total exposure time during the work life of the individual.

The six cases described by Dr. Shapiro have certain common features and findings. The men involved were relatively young or middle-aged, between 30 and 43. Therefore, presbycusis is not a factor. All showed a profound unilateral hearing loss of the sensory neural type. The loss is alleged to have followed short-term noise exposure. The operational procedures of the workers involved the use of pneumatic or hammering tools which produced an intense impact noise. In many of these cases there was an unusual orientation and exposure of one ear of the worker to the source, sound. In most instances the work was being performed in a confined space with reverberation present. Two important bits of information are missing in all of these cases, namely, first, a previous audiogram or hearing record of the worker prior to the alleged incident, and second, a record as to the noise exposure of the working area (not only the sound pressure level and the composition of the noise, but peak readings, inasmuch as we are dealing with an impact type of noise).

The impression I get from the description of these cases and the resultant profound hearing losses is that we are dealing with a high intensity sound pressure level, that it is particularly high at the source and near the affected ear, and that the opposite ear escaped any serious injury because of the shielding effect of the head. Dr. Shapiro states that the noise arising from the pneumatic tools as well as the object may reach a sound pressure level of 125 db., and that it is a broad-band type of noise with most of the energy in the high frequencies, which is more damaging to the ear. While 125 db. may be the quoted sound pressure level produced by some pneumatic tools, the readings will naturally vary with the distance from the source, the nature of the environment, and whether or not an impact meter is used. I, too, have examined several cases of profound unilateral hearing loss following short-term exposure to intense industrial noise. One such case followed the use of a pneumatic chisel and hammer to break up concrete in a confined space; another, the use of a stud gun. Measurement of the noise in the two cases with proper instrumentation revealed sound pressure level readings in excess of 140 db. This is very important, especially when we consider that the men were working in a confined area. I believe it would be worth while for the safety engineers of the insurance carriers who are involved in these cases to measure the peak sound pressure level readings at the source and at various distances away from the source and also to observe the position of the exhaust to the workers' area. This might prove quite important.

Perlman stated that if the sound pressure level is very great with repetitive sounds, the middle ear muscle reflex may be obliterated. If the above opinions should prove to be correct, then these cases really would fall under the grouping of acoustic trauma similar to the intense blasts and explosions and explain the resultant damage.

I agree with Dr. Shapiro that predictive tests which have been formulated to date, based upon producing a temporary threshold shift or "aural overload" are not practical, nor have they been validated in the industrial environment.

I feel that it is most important to have proper preemployment and periodic audiometric tests on workers who are exposed to hazardous noises, blasts, and explosions. Also accurate noise studies should be made of the working environment as described, and ear protective measures used, such as ear plugs, helmets, or treatment of the machines and working areas.

Dr. Shapiro is to be commended for his impartial, objective and scientific approach and particularly for bringing these cases out in the open by presenting them before this otolaryngological group.

DR. JOHN R. LINDSAY: I believe that the first case was a caisson worker and there was a swollen ear drum. Is that correct?

DR. THOMAS C. GALLOWAY: I should like to ask Dr. Shapiro if he has ever had any skeet- or trapshooters in his group.

DR. STANTON FRIEDBERG: What is Dr. Shapiro's opinion as to the possibility of ear injury from telephone clicks?

DR. SHERMAN L. SHAPIRO: In answer to Dr. Lindsay, some of these men worked in caisson foundations but not under compressed air. The first man had had, as I stated, a cold and showed an acute catarrhal otitis when I first saw him, but my tests were not made until after this had subsided.

I have had no personal experience with trapshooters, but Dr. Bunch several years ago examined a number, and some of these did, indeed, show the chief damage on the side next to the gun. I might add that Dr. Bunch years ago also commented how much the noise from an air hammer was amplified in a caisson foundation. He thought it was doubled.

As to our President's question about telephone clicks, I have had some experience with these. They are due to sudden variations in voltage occurring when a connection is made or broken. I have had these sounds analyzed in detail by acoustic physicists; their duration is in milliseconds, and peak intensity, under conditions simulating ordinary use, is between 98 and 115 db. Oddly enough, I just had a woman who alleged deafness from this cause. She was found to be malingering, and that or exaggeration of a preexisting condition has been my diagnosis in the cases which I have seen. I believe that this type of sound can be disagreeable, but I do not believe that it can cause permanent hearing loss.

Abstracts from Current Literature

Ear

THE USEFULNESS OF THE BINAURAL HEARING AID. RAYMOND CARHART, J. Speech & Hearing Disorders 23:42-51 (Feb.) 1958.

Binaural fittings can bring enough added improvement to many persons with mild and moderate hearing losses so that their problems in everyday hearing are greatly simplified.

PALMER, Wichita, Kan.

UNILATERAL DEAFNESS. ALFRED T. LIEBERMAN, Laryngoscope 67:1237 (Dec.) 1957.

The article concerns 57 cases of unilateral deafness of obscure origin that were personally encountered in the past nine years. Causes such as otitis media, trauma, neoplasm, mumps, and other obvious lesions are excluded. Otoscopic examination was normal in all cases. There is a comprehensive review of the literature of unilateral deafness.

The total of 57 cases under study represents 3.8% of all the cases (1475) examined during the nine-year period. Although almost all ages are represented, most cases occurred between 30 and 50. The sexes were approximately equal, as were the right and left sides. Thirty-four were of the conductive and twenty-three of the perceptive type. An analysis is made of each of these types. The conductive type showed (briefly) the following: Tinnitus was present in a little over half the cases, vertigo almost absent. Most cases were between 20 and 50 years of age and had typical audiograms. More than a third had a positive family history of deafness. The etiology of these cases was presumed to be otosclerosis. The 23 nerve type cases showed no such uniformity of symptoms and findings. Some had sudden onset; some were progressive; some followed acute febrile episodes. The etiology of these cases is varied, including toxic neuritis, congenital abnormalities, labyrinthine hydrops, and atrophy of the organ of Corti.

HITSCHLER, Philadelphia.

SCOPE OF SURGERY IN MANAGEMENT OF DEAFNESS. I. B. THORBURN, Brit. M. J. 1:200 (Jan. 25) 1958.

The last three decades have witnessed great progress in the surgical management of conductive deafness with the development of the techniques of fenestration, stapes mobilization, and tympanoplasty. The success that Lempert experienced in devising a one-stage fenestration operation was due to his development of a technique for the treatment of chronic otitis media. He introduced precision surgery, using new instrumentation under the control of binocular loupe magnification. The development of the operating microscope has enabled most otologists to improve their technique and their results with the fenestration operation.

The improved results of Thorburn's second 100 fenestrations as compared with his first 100 cases demonstrate the value of his improved technique. In his first 100 cases, 51 patients achieved hearing of 30 db. or better, which constituted complete patient rehabilitation; 24 had useful improvement of hearing; 22 were worse or not improved; and in 3 cases no follow-up was available. In the second 100 cases, 85 achieved complete rehabilitation; 13 achieved useful improvement of hearing, whereas only 1 was either not improved or was worse, and in 1 case no follow-up was available. These figures were influenced, however, not only by the improving technique but by better selection of favorable cases.

Stapes mobilization has served to provide a second surgical approach to the treatment of otosclerosis which has the advantage that it requires little or no aftercare. Although it gives good results in a smaller percentage of cases than the fenestration operation, it does in its successful cases give very good results. If the hearing gain is lost, the operation can be repeated; or, if it fails, fenestration can be successfully carried out with the added advantage of the knowledge that the stapes footplate is firmly ankylosed. When mobilization fails, owing to fracture of the crura, it is unwise to submit the labyrinth to the risk of trauma by fenestration of the fixed footplate of the stapes.

Tympanoplasty is the most important development in functional otology since Lempert's description of his one-stage fenestration operation. Zöllner and Wullstein worked out the principles of tympanoplasty, and, unless these fundamental principles are followed, tympano-

plasty may be prematurely discredited. The first principle is precision microsurgical technique. This requires the use of an operating microscope, giving from 4 to 12 times magnification, for all work in and around the tympanum. Hemostasis is essential.

The second principle is that pathological changes in the tympanum can be assessed as irreversible or reversible under the operating microscope. Irreversible changes, which include cholesteatoma, keratinizing squamous epithelium, granulations, or osteitis, must be completely removed. Reversible disease, which refers mainly to mucosal swelling, will often resolve. This will provide later a mobile tympanic membrane and mucous membrane covering required for the successful tympanoplasty over most of the medial tympanic wall.

The third principle is that antibiotic therapy helps considerably to control chronic infection, provided irreversible pathology has been eliminated. The antibiotics are used mainly to limit the risk of complications resulting from local operative trauma.

The fourth principle is that full-thickness skin grafts provide a satisfactory medium for the plastic reconstruction of a new tympanic cavity. Postaural skin is especially suitable for full-thickness skin grafting in reconstructing the tympanum, placed over a bed prepared by removing the outer epithelial layer from the remains of the drum and the skin from the adjacent meatal wall. Absorbent gelatin sponge is used to support the graft and to prevent the obliteration of the tympanum during healing and to allow the mucous membrane to line the undersurface of the graft, where it is to form a functioning tympanic membrane. A pedicled meatal skin flap is preferable, however, for use as a lining of the mastoid cavtiy.

The fifth principle is that tympanoplasty attempts to apply the basic audiological principles of leverage and phase differential to achieve the optimum transmission of sound waves to the labyrinth. The new tympanum must be air-containing, and in continuity with a patent Eustachian tube. Cochlear function must be adequate to benefit from an improved conductive mechanism. The functional integrity of the ossicles and of the oval and round windows should be tested. The type of reconstruction will depend upon the result of these tests. Where it is impossible to obtain leverage through complete destruction of the ossicles, the round window must be protected by making a closed tympanum, either normal-sized, or through creation of a small middle ear in continuity with the Eustachian tube. The author states that the hearing results in his cases of tympanoplasty have been very encouraging but that more time must elapse before he feels justified in publishing his results.

JENNES, Waterbury, Conn.

OTOSCLEROSIS: A TWENTY-YEAR RETROSPECT. I. SIMPSON HALL, Proc. Roy. Soc. Med. 51:57 (Jan.) 1958.

A brief historical review of the fenestration operation includes mention of the multistage operation of Sourdille, the early use of the operating microscope by Holmgren, which the author first witnessed in Stockholm in 1938, and the introduction of the single-stage procedure of Lempert. In Hall's first successful case, which still retains the original improvement in hearing, he operated with the multistage technique in 1936 and 1937.

In discussing the pathology of otosclerosis, Hall theorizes as to the etiolcgy of the disease. It is noted that fragilitas ossium has many characteristics in common with otosclerosis and that between 40% and 60% of all cases of fragilitas ossium suffer from otosclerosis. In fragilitas ossium the onset of deafness occurs with healing of bone, suggesting that the healing of bone and the deafness are concurrent. Otosclerosis may therefore be viewed as merely an instance of healing bone. There is a fundamental embryological abnormality of the bone similar, perhaps, to the lack of osteoblastic tissue which is found in fragilitas ossium, which in its healing after the influence of some secondary agent produces what is called otosclerosis.

Anesthesia for the fenestration operation includes induction with thiopentone, with or without a relaxant, followed by intubation and the administration of gas-oxygen. Induced hypotension is utilized, with systolic blood pressure reduced to between 60 and 80 mm. The surgeon performing the fenestration operation must be carefully trained. There is no place for the occasional fenestrator. There is no substitute for thorough cadaver surgery.

One thousand consecutive operations have been analyzed. A successful operation is defined as fulfilling two requirements: a significant improvement as shown by the audiogram and a conviction on the part of the patient that real benefit has resulted from the operation. Achievement of social adequacy is not considered necessary for success. The patient is not rejected for operation because he cannot reach the social adequacy line.

Patients are divided into three groups: (1) ideal cases; (2) poor cases; (3) those between the two extremes. There is also a fourth group which was experimental and which was necessary only in the early stages of the operation. Patients have been divided into groups on a time basis, showing the various types of operations which have been employed. The groups are few, because only one major change in method has been made at a time, and it required about two years to obtain a statistically valid series, with another two years required before that group could be assessed properly. Of the 78 cases operated on by the "improved technique," 63 cases were able to be reviewed, and of these 71% were successful. Of the 189 cases operated on by the cartilage implant technique, 162 were reviewed, and of these 65% were successful. Of the 298 operations performed by the "contact technique," 271 cases were reviewed, and of these 82% were successful. In the 440 cases in which controlled hypotension was utilized, 396 cases were available for review, and of these 90% were improved. All in all, some 20% of the 1000 cases were not available for study, as they could not be traced. In an additional analysis of cases operated on from 1942 to 1947, which allowed time for a 10 years' review, 82% experienced immediate improvement; 78% maintained two-year improvement, and 74% have maintained improvement for 10 years. Of the 59 revisions performed, 14 could not be traced, and of the remainder 71% were successful.

The details of the operative technique used are unfortunately not available in the article as they were shown in a film. The author, however, states that he has followed Sourdille's technique, using either a postauricular or an endaural incision. He describes his present method of cutting the opening in the labyrinth as a technique developed in Edinburgh which is original in so far as he is aware. Hall is distressed by the high incidence of a persistently discharging cavity, representing 13% of his cases, and the lifelong disability which the fenestration operation produces, in terms of the continued need for otologic care and the restriction on certain activities which must be observed by the patient.

JENNES, Waterbury, Conn.

FURTHER INVESTIGATIONS INTO A PECULIARITY OF NORMAL DIRECTIONAL HEARING. L. B. W. JONGKEES and R. A. v. d. VEER, Acta otol-laryng. 49:47 (Jan.-Feb.) 1958.

These experiments were conducted on sitting patients and on those in the horizontal position, on the couch. With these experiments there was no perfect correspondence between objective and subjective sound location on the horizontal plane. A graph made for subjective locating produced an "S" curve. This form was not modified by changing the position of the patient, or by varying the position of the head or the method of indication. When white noise (a complex sound) was used, the curve approached but did not reach the ideal straight line. When tubes were placed down the external auditory canal to eliminate the function of the auricles, the result was unchanged.

LEWY, Chicago.

ARSENIOUS ACID AND THE HEARING ORGAN. GEORGE KELEMEN, JULIAN L. AMBRUS, and CLARA M. AMBRUS, Acta oto-laryng. 49:53 (Jan.-Feb.) 1958.

For a considerable time arsenic and its compounds have been known to act as chemical poisons to the organ of hearing. In the early experiments Blau studied inner ear changes and Ehrlich in 1907 produced waltzing in mice. However, it is almost impossible to obtain laboratory animals free from spontaneous infection in the upper airways and the ear. This study was therefore directed to project the acute picture caused by arsenic against the presumably well-analyzed background of older organizing infection.

Administration of arsenious acid in the rat produced a hemorrhagic reaction. This was followed by an overshadowing hyperemia, but after 48 hours resorption of the free blood was largely accomplished. Generalized suppurative reaction in the middle ear followed quickly thereafter. Bilaterality of the reaction was evidence that it resulted from the arsenic. In the inner ear the initial hemorrhage was quickly absorbed. Suppurative labyrinthitis never developed. It must be assumed that the inner ear situation was decisively influenced by both old and recent middle ear processes. It is not proper to judge conditions in the inner ear without consideration of the condition of the middle ear. This process therefore offers a method for discriminating between good and bad material from the animal farm.

LEWY, Chicago.

PERVERTED NYSTAGMUS—A CLINICAL AND HISTOPATHOLOGIC STUDY. J. S. RIESCO-MACCLURE, and ALFONSO MARTINEZ, Rev. otorrinolaring. 17:1 (June) 1957.

Perverted nystagmus is always a central lesion, but its exact localization has not always been clearly established. Since the authors have found perverted nystagmus in proved lesions of the brain stem, this suggested to them that these lesions are of a partially destructive nature and interfere with some of the pathways coursing through the floor of the 4th ventricle. They base these deductions on Cramner's experiments.

They present a patient who showed post-caloric perverted nystagmus, in whom they found a metastatic nodule in the floor of the fourth ventricle. The original focus was a carcinoma of the thyroid. In view of these postmortem findings, the theory that perverted nystagmus is an evidence of a partially destructive lesion of the fourth ventricle is substantiated.

PERSKY, Philadelphia.

RECRUITMENT AND THE DIFFERENCE LIMEN TEST. J. S. RIESCO-MACCLURE, Rev. otorrinolaring. 17:6 (June) 1957.

The author made a comparative study of recruitment and the difference limen test in 66 patients with different types of hearing loss. Recruitment is usually present in cases of neural deafness with a lesion in the organ of Corti, and is evidenced by an increase in hearing acuity with an increase of the intensity of the tone, so that the impaired ear approximates the better or normal ear in perception. The difference limen test is the amount of intensity change necessary to produce a perceptible difference in loudness. There are two methods—that of Luscher and Zwislocki and that of Denes and Naunton. The author's findings are similar to those of Luscher. In normal ears, the D. L. varies between 1.0 and 1.5 db. In pathologic ears the D. L. is less than 0.8 db. From the diagnostic standpoint, the D. L. test has only a relative value in localizing a lesion, but its findings closely approximate recruitment and can be used with it to determine whether the lesion is cochlear, retrocochlear, or in an indeterminable area.

PERSKY, Philadelphia.

SEROUS OTITIS. RAUL VELASCO, Rev. otorrinolaring. 17:14 (June) 1957.

The author reviewed 54 cases of serous otitis, of which 34 followed a mild coryza, 10 were due to no apparent etiology, 7 due to a severe upper respiratory infection, and 3, aero-otitis.

It is difficult to determine whether this is a secretory condition or a hydrotympanum. Apparently there is an individual symptomology, and it is not the first stage of an acute suppurative process. The etiology is often confusing, since tubal obstruction was not always present, nor was allergy a frequent finding. The exudate was usually sterile, and did not contain eosinophiles.

The treatment recommended was paracentesis plus suction of the secretions from the middle ear, and inflation of the Eustachian tube.

PERSKY, Philadelphia.

IS AURAL VERTIGO EXCLUSIVELY OF PERIPHERAL ORIGIN? J. S. RIESCO-MACCLURE, Rev. otorrinolaring. 17:42 (Dec.) 1957.

To answer the question, Riesco has grouped aural vertigo in the three common clinical forms—postural vertigo, pseudo Ménière's, and Ménière's syndrome.

Postural vertigo is of two types—(1) where the nystagmus changes its direction with a change of the position of the head, and (2) where the nystagmus is in the same direction irrespective of the position of the head. The vertigo is transient, may occur at any time of the day, and usually lasts 10 to 20 seconds. The cause is undetermined, and has been blamed on dental infections, gastrointestinal disturbances, emotional upsets, or post-cranial traumatism. When it has a paroxysmal character, it is benign and does not have any intracranial lesion, and is probably due to a disturbance of the otolithic apparatus. The mechanism is that there is an abnormal or irritative activity of the bulbar vestibular nuclei which produces the paroxysmal symptom. These symptoms can be controlled by Belladenal or Bellargol.

Pseudo Ménière's syndrome is an aural vertigo but without acoustic symptoms. There may be an objective or a subjective vertigo, with a loss of equilibrium, nausea, vomiting, marked chilliness, pallor, and sweating, and it may last 10 minutes to two hours. The author surmises that this syndrome may be of central origin. He cites a case which was due to epilepsy and which responded to anticonvulsive therapy.

True Ménière's syndrome has been defined as vertigo associated with tinnitus and impairment of hearing. The auditory characteristics are (1) a neural deafness with an ascending

curve, (2) the deafness fluctuating from one examination to another, (3) a positive recruitment; (4) a shortening of the difference limen, and (5) diplacusis. The etiology may be that of an autonomic dysfunction of the vascular system which produces hydrops. This may be on a physical, allergic, or emotional basis. There occurs both vasoconstriction and vasodilation, which can produce anoxia in the tissues, with a liberation of toxic substances, such as histamins. There is also an increase in the permeability of the blood vessels which produce an interstitial edema. Consequently, there are two problems—(1) to correct the tissue reactions that were produced by the spastic atonic vascular reactions, and (2) to stabilize the autonomic nervous system. The therapy suggested includes nicotinic acid, histamine desensitization, ammonium chloride, thyroid extract, estrogens in women, and mild sedatives.

The author states that the localization of lesions to account for postural vertigo, pseudo Ménière's and Ménière's complex is most difficult. While some lesions may be of a peripheral nature, a central lesion may produce characteristics of paroxysmal vertigo. The cerebral symptoms that were present in some of his cases were profound sleep, loss of consciousness, mental torpitude, marked falling, and incontinence of urine.

In summarizing his therapeutic measures for aural vertigo, the author divides his cases into three groups—(1) histamine according to the Derlacki technique; (2) nicotinic acid, at times supplemented by vitamin B complex and Belladenal, and (3) anti-convulsive drugs, such as Mesantoin.

PERSKY, Philadelphia.

Larynx

ENDOSCOPIC ASPECTS OF BRONCHOGENIC CARCINOMA. A. J. SEPPALA, Acta oto-laryng. 49:81 (Jan.-Feb.) 1958.

This represents a study of 815 cases of bronchogenic carcinoma. The data follow: 456 were on the right side; 313 were on the left side; 7 were tracheal; 25 involved the carina; 14 cases were bilateral at the hilar region. Half of the lesions were in the upper lobes. Positive biopsies were obtained in 69%. Interestingly, in 67 cases with a negative biopsy cytological examination of the sputum was positive. In about one-half of the cases there was bronchoscopic evidence suggesting carcinoma. The bronchoscopic result was entirely negative in 14% of all the cases.

The histological distribution was as follows: 48% squamous-cell carcinoma; 30% large-cell carcinoma; 13% small-cell carcinoma; 9% adenocarcinoma. Cases considered inoperable at bronchoscopy represented 25%. The incidence, (52%) was highest in the group of small-cell carcinomas. Paralysis of the recurrent laryngeal nerve was present in 63 cases (8%). This indicates the desirability of endoscopic examination in all such cases where the cause of the paralysis is not established.

LEWY, Chicago.

Nose

MALIGNANT TUMORS OF THE NASO-PHARYNX. T. J. MOLONY, Laryngoscope 67:1297 (Dec.) 1957.

Except for China, where it is apparently extremely high, the incidence of malignancy of the nasopharynx is but 0.3% of all body malignancies. The ratio of men to women is seven to one. Most are carcinoma of various types. Symptoms may be neuro-ophthalmic (pain over first division of the fifth nerve, paralysis of the sixth nerve), glandular (metastatic under the sternocleidomastoid muscle), nasopharyngeal (nasal obstruction or hemorrhage), or otological (from blockage of the invasion of the Eustachian tube). The glandular were the commonest presenting symptoms, and the nasopharyngeal next.

A careful and thorough examination of the nasopharynx, impossible with the nasopharyngoscope alone, is stressed.

Treatment consists of radiation. Indications seem to be that cobalt^{60} may be superior to the usual therapy. Survival rate for five years was 9.1%.

HITSCHLER, Philadelphia.

EXPERIENCES IN THE TREATMENT OF THE ALLERGIC NASAL POLYP BY THE INTRAPOLYP INJECTION OF PREDNISOLONE T. B. A. DAVID MYERS, Laryngoscope 68:1 (Jan.) 1958.

Repeated injections of prednisolone T. B. A. directly into nasal polyps caused them to decrease in size, and in many cases to disappear altogether. In some cases there were no recur-

rences. One cubic centimeter of prednisolone T. B. A. (containing 20 mg.) is injected, a little into each polyp, in each nostril. No anesthesia is necessary. It is repeated every five to seven days. In 30 patients, 220 injections were made, an average of 7+ per patient. If there is recurrence, the treatment may be repeated. No reactions or other harmful effects were noted. Surgery of course may be used in conjunction with this tratment.

HITSCHLER, Philadelphia.

Pharynx

PHARYNGEAL LYMPHOID TISSUE IN CHILDREN: ROENTGEN THERAPY OF COMPLICATIONS RESULTING FROM CHRONIC INFECTION. ANTHONY F. ROSSITTO, Eye, Ear, Nose & Throat Month. 36:672 (Nov.) 1957.

Roentgen therapy has been used in more than 6500 children in a series covering a period from 1937 to date. This therapy was first used in patients in whom the symptoms recurred or persisted despite adenoidectomy and tonsillectomy. As experience increased, roentgen therapy became the treatment of choice, irrespective of the presence or absence of tonsils, allergic rhinitis, or sinusitis. This therapy was used in diffuse infections involving the tonsils, adenoids, lymphoid tissue of the pharynx, and nasopharynx, and the Eustachian tube and the adjoining lymph glands, and had usually been preceded by the unsuccessful trial of other methods of treatment. The clinical entity described by the author starts as an acute infection invading the throat and pharyngeal tissues, associated with either poor resistance on the part of the patient, inadequate medical treatment, or resistant infecting organisms. Repeated exacerbations due to some external irritant, rather than a new infection, result in chronic persistent infection of the lymphoid tissues.

Good results were obtained in most patients with one course of treatment, which consisted of roentgen therapy administered over the nasopharynx with additional parts to other areas according to the location of the diseased tissue and symptoms. The roentgen dose to any one part usually represented 1/10 to ⅛ of its skin erythema dose, and the treatments were administered five to seven times at 7- to 10-day intervals. The accumulated dose usually was kept below the erythema dose factor. A second course was indicated only when the leukocyte count did not return to normal, or when symptoms and disease persisted following a reinfection of the pharynx, unrelated to the process for which roentgen therapy was first administered. Although Rossitto can offer no scientific explanation for the favorable results obtained by roentgen therapy, he offers a theory that chronically infected tissue has an overabundance of lymphocytes, leukocytes, and eosinophils, which are destroyed readily when subjected to small doses of x-radiation, with resultant liberation of antibodies and other protective substances.

Roentgen therapy is used to treat infected lymphoid tissue and is not used to remove or treat tonsils per se. It should be administered as early as possible to any child with recurrent infection in the nose or throat, with or without the presence of tonsils, in whom all symptoms, both local and general, return with each attack and in whom all therapeutic measures have failed to prevent exacerbations. The severer this infection, the more rapid and spectacular the results. Trial of x-ray irradiation to the mediastinal and hilar areas has proved very wise in chronic unresolved pneumonitis, which often accompanies mediastinal and hilar glandular adenopathy with previously unexplained elevated temperatures, cough, and chronic asthma. All patients with asthma receive fields of irradiation to the pharyngeal as well as to the mediastinal and hilar areas. Patients with persistent cough can benefit from the use of x-ray irradiation to the pharynx and to the chest in a similar fashion to its effectiveness in persistent cough following measles or whooping cough. A high percentage of permanent cures and good results attests to the use of x-ray therapy as a sound method of treatment, and these opinions are based on visible restoration of the throat tissues to normal or near normal appearance, noticeable and clear-cut permanent remission of the disease and symptoms with restoration of the patient to well-being, associated with grateful acknowledgement by the parents and referring physician for the results obtained. Practically all irradiation failures can be attributed to an accompanying high degree of allergy.

ABSTRACTER'S COMMENT: The enthusiasm of the author, a radiologist, is to be commended. However, it tends to obscure the lack of clinical evidence without which the reader is left more curious than convinced. Since it is difficult to present a scientific evaluation in a subject such as this, the inclusion of detailed case histories might have served to support the author's conclusions.

JENNES, Waterbury, Conn.

Miscellaneous

THE ABSORPTION OF TOPICALLY APPLIED TETRACAINE AND COCAINE. JOHN ADRIANI and DONOVAN CAMPBELL, Laryngoscope 58:65 (Jan.) 1958.

The dangers inherent in the use of local anesthetics is great. Fatalities are frequent.

This study concerns the absorption of the two widely used local anesthetics, tetracaine and cocaine when applied to the mucous membranes of the upper respiratory tract. The absorption is rapid, nearly as rapid as when introduced by vein and much faster than when injected subcutaneously. Likewise the blood levels were much higher than those obtained by subcutaneous infiltration. Addition of epinephrine or use of more concentrated cocaine failed to reduce the absorption. It was found that the absorption also depended on the total amount or weight of the drug applied rather than on the concentration of the solution used.

Tetracaine is 10 times and cocaine 5 times as potent (and toxic) as is procaine, which does not have topical action. As 1 gm. of procaine (infiltrated) is the maximum dose recommended, 100 mg. of tetracaine would seem to be its maximum. However, since the absorption topically is so much faster than by infiltration, this maximum should be reduced to, say 20 mg. Similar reasoning applies to cocaine. However, no exact answer can be given. Also there are those who are intolerant of the drugs; this is unsatisfactory and may even result in a fatal accident in a sensitive subject.

HITSCHLER, Philadelphia.

ADVANCES IN THE DIAGNOSIS AND TREATMENT OF ACUTE PYOGENIC MENINGITIS. EMMANUEL APPELBAUM and CHARLES ADLER, New York J. Med. 58:204 (Jan. 15) 1958.

This is Part I of a study under the general heading of "Recent Advances in Medicine and Surgery," Robert Turell, M.D., Editor. It is issued from the Bureau of Laboratories, New York City Department of Health.

The treatment of acute pyogenic meningitis has been revolutionized during the past 20 to 25 years—first by the sulfonamides, then by the antibiotics. Common causes of the infection are Meningococcus, Pneumococcus, Streptococcus hemophilus influenzae, Staphylococcus, and miscellaneous unclassified organisms. The otologist used to see many more such cases; they are fewer now for various reasons. In infants one does not see early stiffness of the neck. Herpes is frequent, and a rash which promptly fades. The fulminating form is still often fatal—there is so little time. The bacteremia is overwhelming, and circulatory collapse is imminent. Necropsy shows massive hemorrhage into the adrenals. Laboratory studies must include blood smears, blood cultures, spinal fluid analysis (protein is increased). Gram-negative extracellular organisms are often found. Sulfonamides have reduced the mortality markedly. The management calls for plenty of fluids and alkalinization. The sulfadiazine blood level must be maintained, but it demands a careful estimate how much and how long to continue the administration. Adrenal cortical agents are used with some satisfaction even in the fulminating cases. On admission the patient is given an intravenous injection of 1000 to 1500 cc. saline and 5% dextrose plus 100 to 200 mg. hydrocortisone. Repeat in 12 hours. Whole blood transfusions may be of service. Complications are deafness, arthritis, and uveitis. While deafness may be due to destruction of the auditory nerve (commonly in earlier days), it may also be due to the use of antibiotics. In trying to save a life one cannot dwell on eventualities. We have no treatment for this kind of deafness, but we must make sure that it cannot be relieved by operation on labyrinth or brain—abscess for example. These patients are so ill that surgery of any kind must be regarded as "heroic."

The author reports 58 patients with meningococcic meningitis resulting in 56 recoveries and 2 deaths! This is a remarkable showing in the light of past experiences, decades ago. Penicillin is an adjuvant to the sulfonamides. "The antibiotics are particularly valuable in the treatment of patients who are hypersensitive to sulfonamides or have developed a sulfonamide intoxication."

ABSTRACTER'S COMMENT: This essay is of particular interest because in 1926 my wife died (within 24 hours) of an acute fulminating meningitis due to Pneumococcus Type IV, following an acute double otitis media. The infection did not invade the mastoid but went directly to the meninges. In 1958 we have an enormous "advance" against this dreadful disease.

I. W. VOORHEES, New York.

HEREDITY AND ALLERGY. HOWARD G. RAPAPORT, New York J. Med. 58:393 (Feb. 1) 1958.

This is another in the series called "The Fundamentals of Modern Allergy" under the editorship of Samuel J. Prigal, M.D. The battle has waged pro and con for many years. A straddle view is that allergy may be inherited or acquired! One could assume that the hereditary type might be the most refractory and stubborn. The acquired type might be "cured" or rendered innocuous by change of occupation or climate, plus medical measures commonly in use. The discussion is more or less academic, for all the patient wants is relief. He cares little for our discussive attitude toward his affliction. This reminds us of the famous saying of Oliver Wendell Holmes, M.D., when the bitter feud as to the origin or credit concerning etherization was full blown. There is a statue on Boston Common memorializing the fact of anesthesia. Should credit be given to Morton or to Wells? The whimsical author said, "Why not dedicate the monument to E(i)ther?"

We are informed that the present study by Dr. Rapaport was carried out at the Pediatric Clinic of Mount Sinai Hospital, New York City. Also that the entire series will be brought out in book form by Blakiston Company. Date?

I. W. VOORHEES, New York.

THE TREATMENT OF VERTIGO WITH ACETAZOLAMIDE. YVES TARLÉ, Ann. oto-laryng. 74:986 (Dec.) 1957.

The author presents a case of severe Ménière's disease that had not responded to the usual therapeutic measures. Largactal (chlorpromazine) was given in increasing doses until the patient was taking 40 drops a day in divided doses. While this medication seemed to give relief of symptoms, any attempt of reduction or withdrawal of the drug produced a recurrence of the symptoms. Acetazolamide (250 mg. daily) was added to the treatment. The dosage of the Largactal was reduced to 10 drops daily. The patient was completely controlled by this routine.

The rationale of treatment was that the Largactal decreases the vestibular reflectivity, since 80% of Ménière's cases are vagotonics. On the other hand, the action of acetazolamide is directed to the hydrodynamics of the cerebrum. This produces an increased elimination of potassium and a subsequent fluid reduction. Further observation is warranted for his combined therapy.

PERSKY, Philadelphia.

Books

Les Sinusites de l'enfance. By J. Terracol and Y. Guerrier. Price, 1400 fr. Pp. 166, with 28 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris 6e, 1958.

This monograph presents a detailed and meticulous discussion of the problem of sinusitis in children. It begins with an embryologic and anatomic description of the various sinuses. The etiologic factors are presented, indicating that the usual causes are infection and allergy. The commonest sinuses involved are the maxillary and ethmoid sinuses. Other factors may be heredity, lymphatic and neuroarthritic diatheses, endocrine deficiencies, and metabolic disorders.

The symptomatology of sinusitis in infancy differs from that in adults. Acute sinusitis is evidenced by (1) acute osteomyelitis of the superior maxilla, a rare condition; (2) acute ethmoiditis of either the anterior or the posterior cells; (3) acute maxillary sinusitis. Chronic sinusitis may involve the frontal, maxillary, or ethmoid sinuses.

Complications may be orbital, cranial, or intracranial, thrombophlebitis, meningitis, respiratory involvement, circulatory and digestive disturbances.

Treatment of the acute stages is usually medical—to maintain the ventilation of the nose and permit proper drainage. Various forms of instillation sprays and inhalations are recommended. Aerosol therapy is advised in the acute state, while Proetz displacement is recommended for the chronic conditions. Antibiotics, autogenous vaccines, thyrohormonal therapy are used where indicated. The use of steroids has produced dramatic results, particularly in the allergic cases. Surgery is usually reserved for the chronic cases, although it has its indications in the presence of an acute orbital phlegmon.

This excellent monograph is well worth while. There is a detailed summary in English.

Le Cancer de la corde vocale. By J. Piquet. Price, 1600 fr. Pp. 204, with 53 illustrations. Masson & Cie, 120 boulevard Saint-Germain, Paris 6e, 1958.

This monograph presents the problems of cancer of the vocal cords in a concise and logical manner. The discussion is concerned with cancer involving the vocal cords, the floor of the ventricle of Morgagni, and the subglottic area. Among the possible etiologic factors, tobacco is stressed as an important cause. The anatomy, histopathology, and clinical findings are presented in detail and are suitably amplified by descriptive illustrations.

Under treatment, the author discusses the indications and limitations for each of the following procedures: endoscopic denudation of the lesion, with or without galvano-cautery, thyrotomy, hemilaryngectomy, and total laryngectomy. He presents his personal modification of each of the above procedures. The role of roentgen therapy with its indications and limitations is discussed. The final section deals with rehabilitation of the laryngectomized patient.

The monograph also contains a synopsis in English.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.

International Bronchoesophagological Society

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Mexican Society of Otolaryngology

President: Dr. Rafael Giorgana
Secretary: Dr. Carlos Valenzuela, Monterrey 47-201, Mexico 7, D. F.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

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Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
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NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Victor R. Alfaro, Washington, D. C.
Secretary: Dr. Walter E. Heck, San Francisco.
Meeting: Atlantic City, June 8-12, 1959.

American Academy of Ophthalmology and Otolaryngology

President: Dr. LeRoy A. Schall, 243 Charles St., Boston.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Secretary: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3.
Place: The Homestead, Hot Springs, Va. Time: March 10-11, 1959 (afternoon only).

American Laryngological Association

President: Dr. Fred W. Dixon, 2060 E. 9th St., Cleveland.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: The Homestead, Hot Springs, Va. Time: March 8-9, 1959.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: The Homestead, Hot Springs, Va. Time: March 10-12, 1959 (mornings only).

SECTIONS:

Eastern.—Chairman: Dr. Joseph L. Goldman, New York.
Place: The Savoy Plaza, New York. Time: Jan. 8-9, 1959.
Southern.—Chairman: Dr. James T. King, Atlanta, Ga.
Place: Hotel-Atlanta Biltmore, Atlanta, Ga. Time: Jan. 23-24, 1959.
Middle.—Chairman: Dr. Alfred J. Cone, St. Louis, Mo.
Place: Park Plaza Hotel, St. Louis. Time: Jan. 12-13, 1959.
Western.—Chairman: Dr. Robert C. McNaught, San Francisco, Calif.
Place: The Ahwahnee Hotel, Yosemite Valley, Calif. Time: Jan. 17-18, 1959.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.

American Otological Society, Inc.

President: Dr. Moses H. Lurie, 483 Beacon St., Boston.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: The Homestead, Hot Springs, Va. Time: March 13-14, 1959.

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President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Joseph G. Gilbert, 75 Barberry Lane, Roslyn Heights, N. Y.

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Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Lt. Col. Stanley H. Bear (MC), USAF, Maxwell AFB, Ala.
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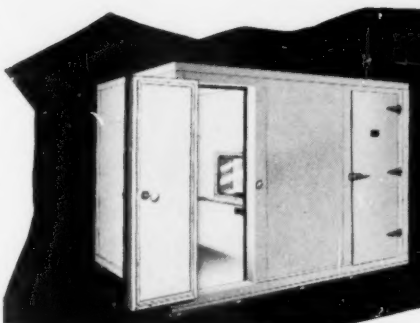

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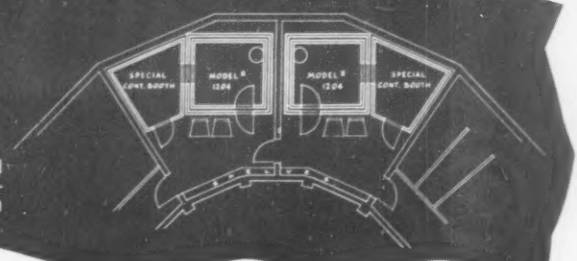

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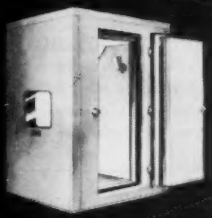

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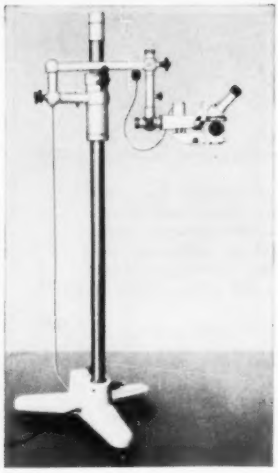

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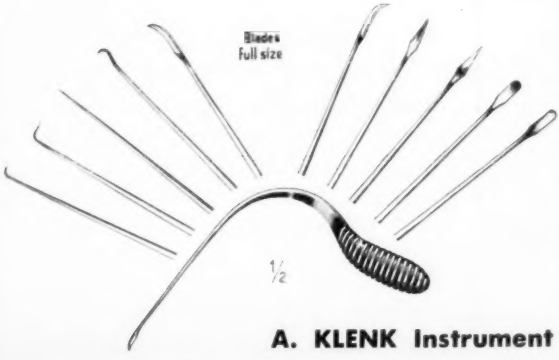

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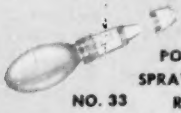

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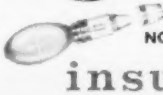

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